

- Cardiology (290)
- Clinical haematology (13)
- Clinical pharmacology (13)
- Clinical sciences (13)
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2600 MCQ with Illustrated Answer
Full Topics Note
14 Chapter

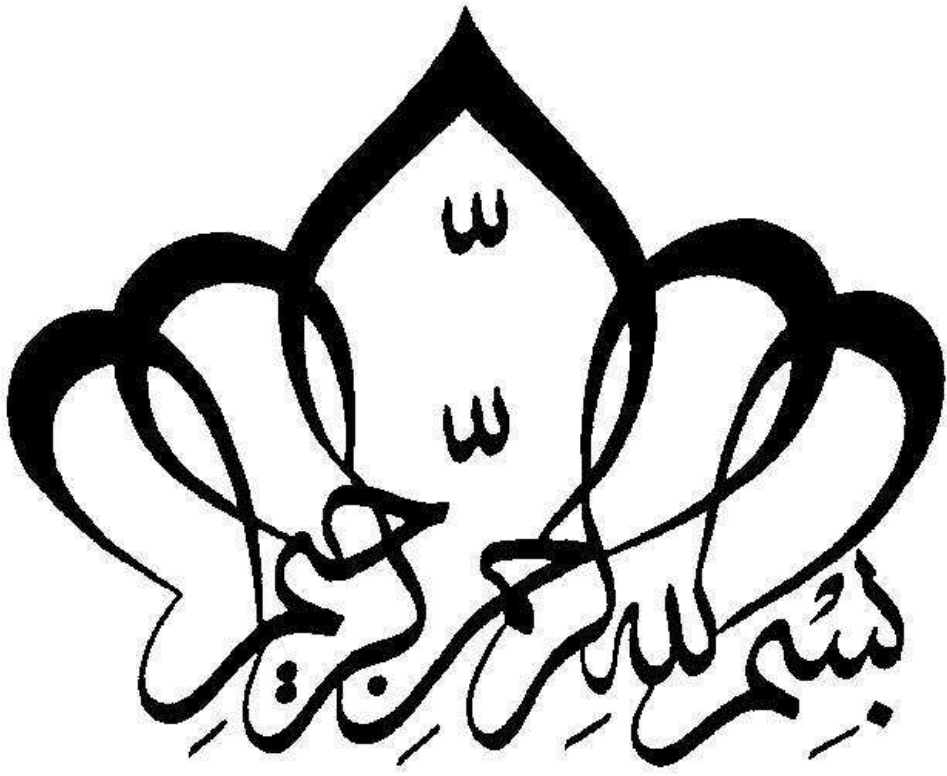
Gastroenterology

P. M MCQ Bank 2017



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قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
۞ إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

إهداء

للكل آية من كتاب الله حفظناها ولكل معلومة قرأناها ونسيناها لعل الله يحمينا من النسيان ويروها إلينا
للكل شخص وعونا له في ظهر الغيب لعل الله أن يستجيب دعائنا
للكل مريض قرأ له ما أله لعل الله يشفيه ويعير إليه صحته وعافيته
للكل لحظة تألم بها أحببنا لعل الله لا يريهم الألم ولا يضييهم مرة أخرى
للكل موقف اجتجت به عيناى فلم تلبيانى لعل الله لا يضرنى بهما في حياتى
للكل حالم صاحب أهراق ومباوى لعل الله يحقق له مقاصده حتى يرضيه ويرضى عنه
للكل مجتهد لعل الله يجعل له من التوفيق أوفر حظ ونصيب ويهون له الأسباب كلها من حيث لا يدري من غير ضراء مضرة ولا فتنة مضلة
للكل من فقر شيئا في الحياة لعل الله يعيره إليه أو يعوضه بما يسعده
للكل شخص ابتعدنا عنه لعل الله يجمعه بمن هم أفضل منا
وأخيرا وليس آخرا لأهلنا لعل الله يجعلها لهم صرقة جارية ترو إليهم قليلا من فضلهم علينا وتكون سببا يلم شملنا تحت سقف واحد بعد أن
هجرتنا الحروب والمصائب

يقول أحد الصالحين

إِذَا رَأَيْتَ أَنَّ اللَّهَ وَفَقَكَ لِإِخْوَةِ تَعْيْنِكَ عَلَى الْخَيْرِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ بِكَ خَيْرًا !! وَإِذَا وَفَقَكَ اللَّهَ لِلدُّعَاءِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ أَنْ يُعْطِيَكَ !! وَإِذَا
وَفَقَكَ لِقَضَاءِ حَوَائِجِ النَّاسِ فَأَعْلَمْ أَنَّ لَكَ يَتَخَلَّى عَنْكَ !! وَإِذَا وَفَقَكَ اللَّهَ لِذِكْرِهِ.. فَأَعْلَمْ أَنَّكَ مُحِبٌّ !!.. وَإِذَا أَحْبَبَّكَ أَحَدٌ.. وَنَصَرَكَ.. وَأَيَّرَكَ..
وَأُسْتَجَابَ لِدُعَائِكَ !!

This issue of **MRCPPART1 MADE EASY 2017** contains the following:

- Full topic note.
- Around 2600 MCQs from **PM 2017 BANK** covering 14 chapter in internal medicine
- External links for the related guidelines (such as NICE, SIGN, RCP ...etc).
- Comments with illustrative pictures and links to external media.
- Most Recommended QBank for MRCP Part 1, Arab Board, Jordanian Board and other internal medicine assessment exams.

Please Note that

Each chapter begins with notes section. These notes are repeated under the Questions in the website. In these collections the notes are not repeated under each question except in some to avoid unnecessary repetition. For clarity and convenience the title of the related note is only mentioned after the comments in case the reader want to refer back to the respective note in the notes section.

The illustration below is to show and Example

☐	Minimal change disease
☐	Post-streptococcal glomerulonephritis
☐	Goodpasture's syndrome

Goodpasture's syndrome

- IgG deposits on renal biopsy
- anti-GBM antibodies

• These changes are characteristic of Goodpasture's syndrome.

[Discuss and give feedback](#)

Goodpasture's syndrome 

Comment of the Q

title of the note in case the reader want to refer back to the notes sections

MRCPPART1 MADE EASY 2017

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إعداد وتنسيق

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&

A. MURAD

لا تنسونا من صالح دعائكم

ما كان من خطأ فمن أنفسنا وما كان من توفيق فمن الله

تم بحمد الله وتوفيقه ومنه

جعل الله عملنا متقبلاً خالصاً لوجهه الكريم

Reference ranges

Reference ranges vary according to individual labs. All values are for adults unless otherwise stated

Full blood count

Haemoglobin	Men: 13.5-18 g/dl Women: 11.5-16 g/dl
Mean cell volume	82-100 fl
Platelets	150-400 * 10 ⁹ /l
White blood cells	4-11 * 10 ⁹ /l

Urea and electrolytes

Sodium	135-145 mmol/l
Potassium	3.5 - 5.0 mmol/l
Urea	2.0-7 mmol/l
Creatinine	55-120 umol/l
Bicarbonate	22-28 mmol/l
Chloride	95-105 mmol/l

Liver function tests

Bilirubin	3-17 umol/l
Alanine transferase (ALT)	3-40 iu/l
Aspartate transaminase (AST)	3-30 iu/l
Alkaline phosphatase (ALP)	30-100 umol/l
Gamma glutamyl transferase (yGT)	8-60 u/l
Albumin	35-50 g/l
Total protein	60-80 g/l

Other haematology

Erythrocyte sedimentation rate (ESR)	Men: < (age / 2) mm/hr Women: < ((age + 10) / 2) mm/hr
Prothrombin time (PT)	10-14 secs
Activated partial thromboplastin time (APTT)	25-35 secs
Ferritin	20-230 ng/ml
Vitamin B12	200-900 ng/l
Folate	3.0 nmol/l
Reticulocytes	0.5-1.5%
D-Dimer	< 400 ng/ml

Other biochemistry

Calcium	2.1-2.6 mmol/l
Phosphate	0.8-1.4 mmol/l
CRP	< 10 mg/l
Thyroid stimulating hormone (TSH)	0.5-5.5 mu/l
Free thyroxine (T4)	9-18 pmol/l
Total thyroxine (T4)	70-140 nmol/l
Amylase	70-300 u/l

Uric acid	0.18-0.48 mmol/l
-----------	------------------

Arterial blood gases

pH	7.35 - 7.45
pCO2	4.5 - 6.0 kPa
pO2	10 - 14 kPa

Lipids

Desirable lipid values depend on other risk factors for cardiovascular disease, below is just a guide:

Total cholesterol	< 5 mmol/l
Triglycerides	< 2 mmol/l
HDL cholesterol	> 1 mmol/l
LDL cholesterol	< 3 mmol/l

Chapter: Gastroenterology

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Chapter: Gastroenterology

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Chapter: Gastroenterology

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Chapter: Gastroenterology

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Abdominal pain

The table below gives characteristic exam question features for conditions causing abdominal pain. **Unusual and 'medical' causes of abdominal pain should also be remembered:**

- myocardial infarction
- diabetic ketoacidosis
- pneumonia
- acute intermittent porphyria
- lead poisoning

Condition	Characteristic exam feature
Peptic ulcer disease	<u>Duodenal ulcers</u> : more common than gastric ulcers, epigastric pain relieved by eating Gastric ulcers: epigastric pain worsened by eating Features of upper gastrointestinal haemorrhage may be seen (haematemesis, melena etc)
Appendicitis	Pain initial in the central abdomen before localising to the right iliac fossa Anorexia is common Tachycardia, low-grade pyrexia, tenderness in RIF Rovsing's sign: more pain in RIF than LIF when palpating LIF
Acute pancreatitis	Usually due to alcohol or gallstones Severe epigastric pain Vomiting is common Examination may reveal tenderness, ileus and low-grade fever Periumbilical discolouration (Cullen's sign) and flank discolouration (Grey-Turner's sign) is described but rare
Biliary colic	Pain in the RUQ radiating to the back and interscapular region, may be following a fatty meal. Slight misnomer as the pain may persist for hours Obstructive jaundice may cause pale stools and dark urine It is sometimes taught that patients are female, forties, fat and fair although this is obviously a generalization

Chapter: Gastroenterology

Acute cholecystitis	History of gallstones symptoms (see above) Continuous RUQ pain Fever, raised inflammatory markers and white cells Murphy's sign positive (arrest of inspiration on palpation of the RUQ)
Diverticulitis	Colicky pain typically in the LLQ Fever, raised inflammatory markers and white cells
Abdominal aortic aneurysm	Severe central abdominal pain radiating to the back Presentation may be catastrophic (e.g. Sudden collapse) or sub-acute (persistent severe central abdominal pain with developing shock) Patients may have a history of cardiovascular disease
Intestinal obstruction	History of malignancy/previous operations Vomiting Not opened bowels recently 'Tinkling' bowel sounds

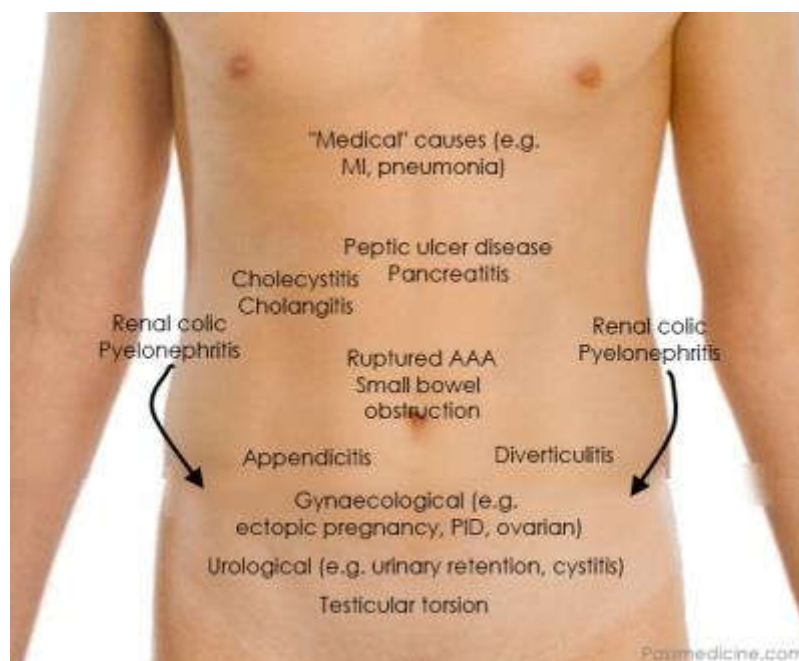


Diagram showing stereotypical areas where particular conditions present. The diagram is not exhaustive and only lists the more common conditions seen in clinical practice. Note how pain from renal causes such as renal/ureteric colic and pyelonephritis may radiate and move from the loins towards the suprapubic area.

Achalasia

Failure of oesophageal peristalsis and of relaxation of lower oesophageal sphincter (LOS) due to degenerative loss of ganglia from Auerbach's plexus i.e. LOS contracted, oesophagus above dilated. Achalasia typically presents in middle-age and is equally common in men and women.

Clinical features:

- dysphagia of BOTH liquids and solids
- typically variation in severity of symptoms
- heartburn
- regurgitation of food – may lead to cough, aspiration pneumonia etc
- malignant change in small number of patients

Investigations:

- manometry: excessive LOS tone which doesn't relax on swallowing - considered most important diagnostic test
- barium swallow shows grossly expanded oesophagus, fluid level, 'bird's beak' appearance
- CXR: wide mediastinum, fluid level

Treatment:

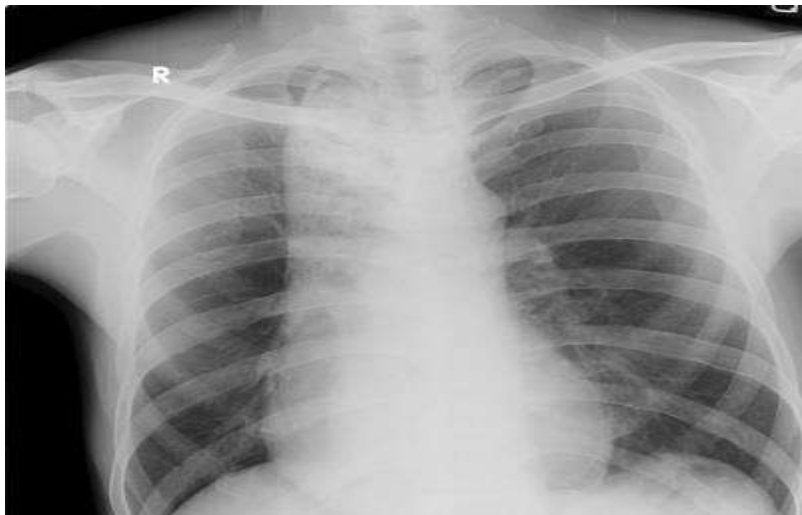
- intra-sphincteric injection of botulinum toxin
- Heller cardiomyotomy
- balloon dilation
- drug therapy has a role but is limited by side-effects



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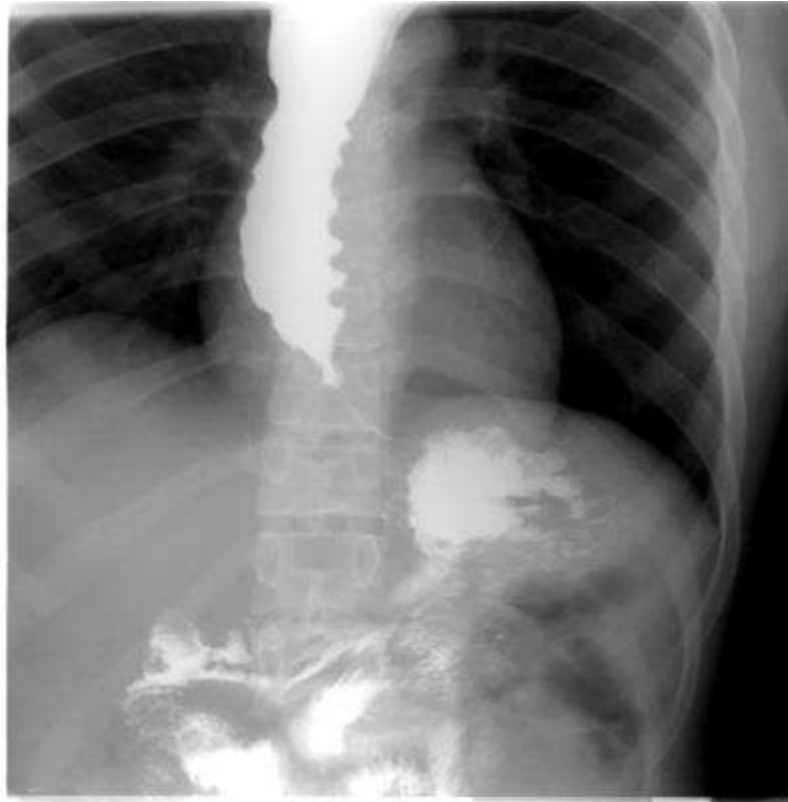
This film demonstrates the classical 'bird's beak' appearance of the lower oesophagus that is seen in achalasia. An air-fluid level is also seen due to a lack of peristalsis



© Image used on license from [Radiopaedia](#)



Mediastinal widening secondary to achalasia. An air-fluid level can sometimes be seen on CXR but it is not visible on this film



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Barium swallow - grossly dilated filled oesophagus with a tight stricture at the gastroesophageal junction resulting in a 'bird's beak' appearance. Tertiary contractions give rise to a corkscrew appearance of the oesophagus

Acute appendicitis

History

- peri-umbilical abdominal pain (visceral stretching of appendix lumen and appendix is mid gut structure) radiating to the right iliac fossa due to localised parietal peritoneal inflammation
- vomit once or twice but marked and persistent vomiting is unusual
- diarrhoea is rare. However, pelvic appendicitis may cause localised rectal irritation of some loose stools. A pelvic abscess may also cause diarrhoea
- mild pyrexia is common - temperature is usually 37.5-38°C. Higher temperatures are more typical of conditions like mesenteric adenitis
- anorexia is very common. It is very unusual for patients with appendicitis to be hungry
- around 50% of patients have the typical symptoms of anorexia, peri-umbilical pain and nausea followed by more localised right lower quadrant pain

Examination

- generalised peritonitis if perforation has occurred or localised peritonism
- retrocaecal appendicitis may have relatively few signs
- digital rectal examination may reveal boggy sensation if pelvic abscess is present, or even tenderness with a pelvic appendix

Diagnosis

- typically raised inflammatory markers coupled with compatible history and examination findings should be enough to justify appendicectomy.
- urine analysis may show mild leucocytosis but no nitrites.
- ultrasound is useful in females where pelvic organ pathology is suspected. Although it is not always possible to visualise the appendix on ultrasound, the presence of free fluid (always pathological in males) should raise suspicion.

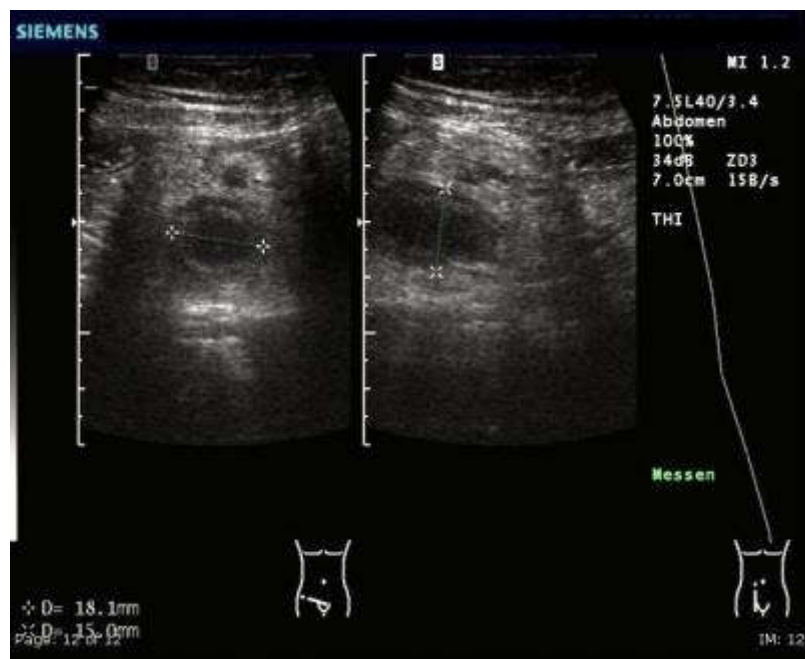


Image sourced from [Wikipedia](#)

Ultrasound examination may show evidence of luminal obstruction and thickening of the appendiceal wall as shown below.

Management

- appendicectomy which can be performed via either an open or laparoscopic approach.
- administration of metronidazole reduces wound infection rates.
- patients with perforated appendicitis require copious abdominal lavage.
- patients without peritonitis who have an appendix mass should receive broad spectrum antibiotics and consideration given to performing an interval appendicectomy.
- be wary in the older patients who may have either an underlying caecal malignancy or perforated sigmoid diverticular disease.

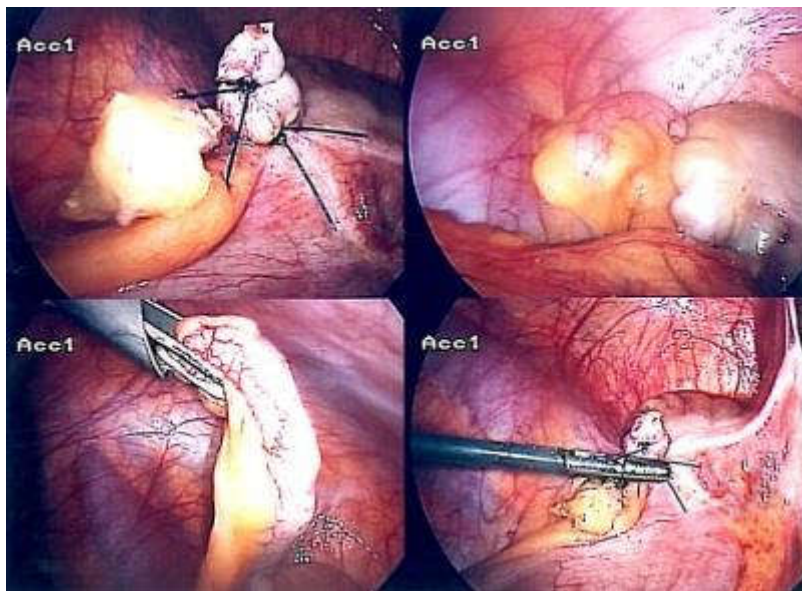


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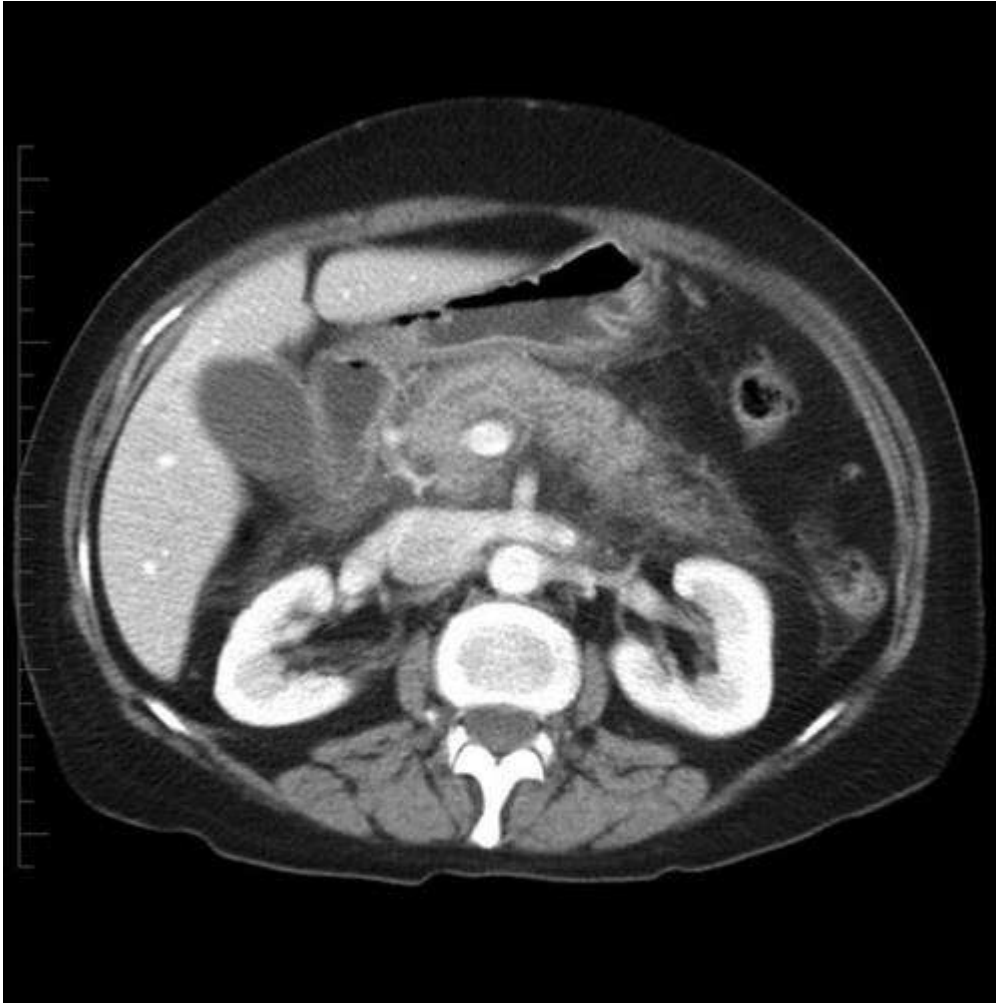
Laparoscopic appendicectomy is becoming increasingly popular as demonstrated below

Acute pancreatitis: causes

The vast majority of cases in the UK are caused by gallstones and alcohol

Popular mnemonic is **GET SMASHED**

- **G**allstones
- **E**thanol
- **T**rauma
- **S**teroids
- **M**umps (other viruses include Cocksackie B)
- **A**utoimmune (e.g. polyarteritis nodosa), *Ascaris* infection
- **S**corpion venom
- **H**ypertriglyceridaemia, **H**yperchylomicronaemia, **H**ypercalcaemia, **H**ypothermia
- **E**RCP
- **D**rugs (azathioprine, mesalazine*, didanosine, bendroflumethiazide, furosemide, pentamidine, steroids, sodium valproate)



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CT from a patient with acute pancreatitis. Note the diffuse parenchymal enlargement with oedema and indistinct margins.

*pancreatitis is 7 times more common in patients taking mesalazine than sulfasalazine

External Links:

[British Society of Gastroenterology](#)

2005 acute pancreatitis guidelines

[Journal of Clinical and Diagnostic Research](#)

APACHE II scoring system for Acute Pancreatitis

Acute pancreatitis: features

Rare features associated with pancreatitis include:

- ischaemic (Purtscher) retinopathy - may cause temporary or permanent blindness

External Links:

[British Society of Gastroenterology](#)

2005 acute pancreatitis guidelines

Acute upper gastrointestinal bleeding

NICE published guidelines in 2012 on the management of acute upper gastrointestinal bleeding which is most commonly due to either peptic ulcer disease or oesophageal varices. Some of the key points are detailed below.

Risk assessment

- use the Blatchford score at first assessment, and
- the full Rockall score after endoscopy

Blatchford score

Admission risk marker	Score
Urea (mmol/l)	6.5 - 8 = 2 8 - 10 = 3 10 - 25 = 4 > 25 = 6
Haemoglobin (g/l)	Men • 12 - 13 = 1 • 10 - 12 = 3 • < 10 = 6 Women • 10 - 12 = 1 • < 10 = 6
Systolic blood pressure (mmHg)	100 - 109 = 1 90 - 99 = 2 < 90 = 3
Other markers	Pulse $\geq 100/\text{min}$ = 1 Presentation with melaena = 1 Presentation with syncope = 2 Hepatic disease = 2 Cardiac failure = 2

Patients with a Blatchford score of **0** may be considered for early discharge

Resuscitation

- ABC, wide-bore intravenous access * 2
- platelet transfusion if actively bleeding platelet count of less than $50 \times 10^9/\text{litre}$
- fresh frozen plasma to patients who have either a fibrinogen level of less than 1 g/litre, or a prothrombin time (international normalised ratio) or activated partial thromboplastin time greater than 1.5 times normal
- prothrombin complex concentrate to patients who are taking warfarin and actively bleeding

Endoscopy

- should be offered immediately after resuscitation in patients with a severe bleed
- all patients should have endoscopy within 24 hours

Management of non-variceal bleeding

- NICE do not recommend the use of proton pump inhibitors (PPIs) before endoscopy to patients with suspected non-variceal upper gastrointestinal bleeding although PPIs should be given to patients with non-variceal upper gastrointestinal bleeding and stigmata of recent haemorrhage shown at endoscopy
- if further bleeding then options include repeat endoscopy, interventional radiology and surgery

Management of variceal bleeding

- terlipressin and prophylactic antibiotics should be given to patients at presentation (i.e. before endoscopy)
- band ligation should be used for oesophageal varices and injections of N-butyl-2-cyanoacrylate for patients with gastric varices
- transjugular intrahepatic portosystemic shunts (TIPS) should be offered if bleeding from varices is not controlled with the above measures

Chapter: Gastroenterology

External Links:

[NICE](#)

2012 Acute upper gastrointestinal bleeding: management

[Royal College of Physicians](#)

2012 Managing acute upper gastrointestinal bleeding in the acute assessment unit

Alkaline phosphatase

Causes of raised alkaline phosphatase (ALP):

- liver: cholestasis, hepatitis, fatty liver, neoplasia
- Paget's
- osteomalacia
- bone metastases
- hyperparathyroidism
- renal failure
- physiological: pregnancy, growing children, healing fractures

The table below splits the causes according to the calcium level:

Raised ALP and raised calcium	Raised ALP and low calcium
• Bone metastases • Hyperparathyroidism	• Osteomalacia • Renal failure

Aminosalicylate drugs

5-aminosalicylic acid (5-ASA) is released in the colon and is not absorbed. It acts locally as an anti-inflammatory. The mechanism of action is not fully understood but 5-ASA may inhibit prostaglandin synthesis

Sulphasalazine:

- a combination of sulphapyridine (a sulphonamide) and 5-ASA
- many side-effects are due to the sulphapyridine moiety: rashes, oligospermia, headache, Heinz body anaemia, megaloblastic anaemia
- other side-effects are common to 5-ASA drugs (see mesalazine)

Mesalazine

- a delayed release form of 5-ASA
- sulphapyridine side-effects seen in patients taking sulphasalazine are avoided
- mesalazine is still however associated with side-effects such as GI upset, headache, agranulocytosis, pancreatitis*, interstitial nephritis

Olsalazine

- two molecules of 5-ASA linked by a diazo bond, which is broken by colonic bacteria

*pancreatitis is 7 times more common in patients taking mesalazine than sulfasalazine

External Links:

[Gut](#)

Adverse effects of sulfasalazine and mesalazine

Amoebiasis

Amoebiasis is caused by *Entamoeba histolytica* (an amoeboid protozoan) and spread by the faecal-oral route. It is estimated that 10% of the world's population is chronically infected. Infection can be asymptomatic, cause mild diarrhoea or severe amoebic dysentery. Amoebiasis also causes liver and colonic abscesses

Amoebic dysentery

- profuse, bloody diarrhoea
- stool microscopy may show trophozoites
- treatment is with metronidazole

Amoebic liver abscess

- usually a single mass in the right lobe (may be multiple)
- features: fever, RUQ pain
- serology positive in > 90%

Anal fissure

Anal fissures are longitudinal or elliptical tears of the squamous lining of the distal anal canal. If present for less than 6 weeks they are defined as acute, and chronic if present for more than 6 weeks. Around 90% of anal fissures occur on the posterior midline

Risk factors

- constipation
- inflammatory bowel disease
- sexually transmitted infections e.g. HIV, syphilis, herpes

Features

- painful, bright red, rectal bleeding

Management of an acute anal fissure (< 6 weeks):

- dietary advice: high-fibre diet with high fluid intake
- bulk-forming laxatives are first line - if not tolerated then lactulose should be tried
- lubricants such as petroleum jelly may be tried before defecation
- topical anaesthetics

-analgesia

- topical steroids do not provide significant relief

Management of a chronic anal fissure (> 6 weeks):

- the above techniques should be continued
- topical glyceryl trinitrate (GTN) is first line treatment for a chronic anal fissure
- if topical GTN is not effective after 8 weeks then secondary referral should be considered for surgery or botulinum toxin

External Links:

[Clinical Knowledge Summaries](#)

Anal fissure guidelines

Ascending cholangitis

Ascending cholangitis is a bacterial infection of the biliary tree. The most common predisposing factor is gallstones.

Charcot's triad of right upper quadrant (RUQ) pain, fever and jaundice occurs in about 20-50% of patients

- fever is the most common feature, seen in 90% of patients
- RUQ pain 70%
- jaundice 60%
- hypotension and confusion are also common

Management

- intravenous antibiotics
 - endoscopic retrograde cholangiopancreatography (ERCP) after 24-48 hours to relieve any obstruction
-

Autoimmune hepatitis

Autoimmune hepatitis is condition of unknown aetiology which is most commonly seen in young females. Recognised associations include other autoimmune disorders, hypergammaglobulinaemia and HLA B8, DR3. Three types of autoimmune hepatitis have been characterised according to the types of circulating antibodies present

Type I	Type II	Type III
Anti-nuclear antibodies (ANA) and/or anti-smooth muscle antibodies (SMA)	Anti-liver/kidney microsomal type 1 antibodies (LKM1)	Soluble liver-kidney antigen
Affects both adults and children	Affects children only	Affects adults in middle-age

Features

- may present with signs of chronic liver disease
- acute hepatitis: fever, jaundice etc (only 25% present in this way)
- amenorrhoea (common)
- ANA/SMA/LKM1 antibodies, raised IgG levels
- liver biopsy: inflammation extending beyond limiting plate 'piecemeal necrosis', bridging necrosis

Management

- steroids, other immunosuppressants e.g. azathioprine
- liver transplantation

Bacterial overgrowth: investigation

The gold standard investigation of bacterial overgrowth is small bowel aspiration and culture

Other possible investigations include:

- hydrogen breath test
- 14C-xylose breath test
- 14C-glycocholate breath test: used increasingly less due to low specificity

In practice many clinicians give an empirical course of antibiotics as a trial

External Links:

[British Society of Gastroenterology](#)

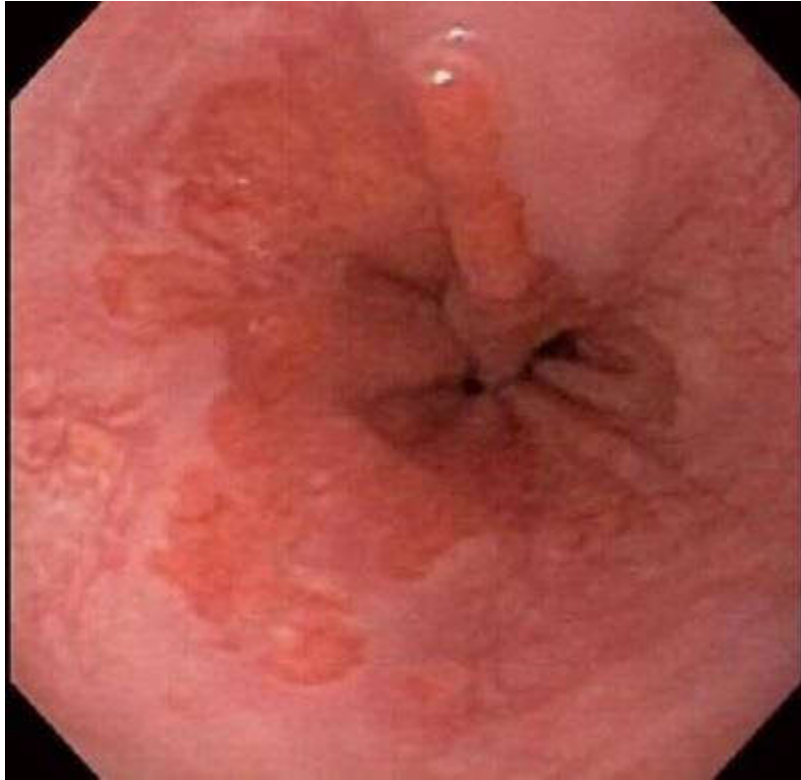
2003 Guidelines for the investigation of chronic diarrhoea

Barrett's oesophagus

Barrett's refers to the metaplasia of the lower oesophageal mucosa, with the usual squamous epithelium being replaced by columnar epithelium. There is an increased risk of oesophageal adenocarcinoma, estimated at 50-100 fold.

Histological features

- the columnar epithelium may resemble that of either the cardiac region of the stomach or that of the small intestine (e.g. with goblet cells, brush border)



Endoscopy image showing a short segment of Barrett's oesophagus

Management

- endoscopic surveillance with biopsies
- high-dose proton pump inhibitor: whilst this is commonly used in patients with Barrett's the evidence base that this reduces the change of progression to dysplasia or induces regression of the lesion is limited

External Links:

[British Society of Gastroenterology](#)

2013 Barrett's oesophagus guidelines

[NICE](#)

2010 Barrett's oesophagus guidelines

Bile-acid malabsorption

le-acid malabsorption is a cause of chronic diarrhoea. This may be primary, due to an excessive production of bile acid, or secondary to an underlying gastrointestinal disorder causing reduced bile acid absorption. Secondary causes are often seen in patients with ileal disease, such as with Crohn's.

Other secondary causes include:

- cholecystectomy
- coeliac disease
- small intestinal bacterial overgrowth

Investigation:

- the test of choice is **SeHCAT**
- nuclear medicine test using a gamma-emitting selenium molecule in selenium homocholic acid taurine or tauroselcholic acid (SeHCAT)
- scans are done 7 days apart to assess the retention/loss of radiolabelled ⁷⁵SeHCAT

Management:

- bile acid sequestrants e.g. cholestyramine

Budd-Chiari syndrome

Budd-Chiari syndrome, or hepatic vein thrombosis, is usually seen in the context of underlying haematological disease or another procoagulant condition

Causes:

- polycythaemia rubra vera
- thrombophilia: activated protein C resistance, antithrombin III deficiency, protein C & S deficiencies
- pregnancy
- oral contraceptive pill.

Features

- abdominal pain: sudden onset, severe
- ascites
- tender hepatomegaly

Child-Pugh classification of liver cirrhosis

The Child-Pugh classification is a scoring system to assess the severity of liver cirrhosis:

Score	1	2	3
Bilirubin ($\mu\text{mol/l}$)	<34	34-50	>50
Albumin (g/l)	>35	28-35	<28
Prothrombin time, prolonged by (s)	<4	4-6	>6
Encephalopathy	none	mild	marked
Ascites	none	mild	marked

Summation of the scores allows the severity to be graded either A, B or C:

- < 7 = A
- 7-9 = B
- > 9 = C

Chronic pancreatitis

Chronic pancreatitis is an inflammatory condition which can ultimately affect both the exocrine and endocrine functions of the pancreas. Around 80% of cases are due to alcohol excess with up to 20% of cases being unexplained

Features:

- pain is typically worse 15 to 30 minutes following a meal
- steatorrhea: symptoms of pancreatic insufficiency usually develop between 5 and 25 years after the onset of pain
- diabetes mellitus develops in the majority of patients. It typically occurs more than 20 years after symptom begin

Investigation:

- abdominal x-ray shows pancreatic calcification in 30% of cases
- CT is more sensitive at detecting pancreatic calcification. Sensitivity is 80%, specificity is 85%
- functional tests: faecal elastase may be used to assess exocrine function if imaging inconclusive

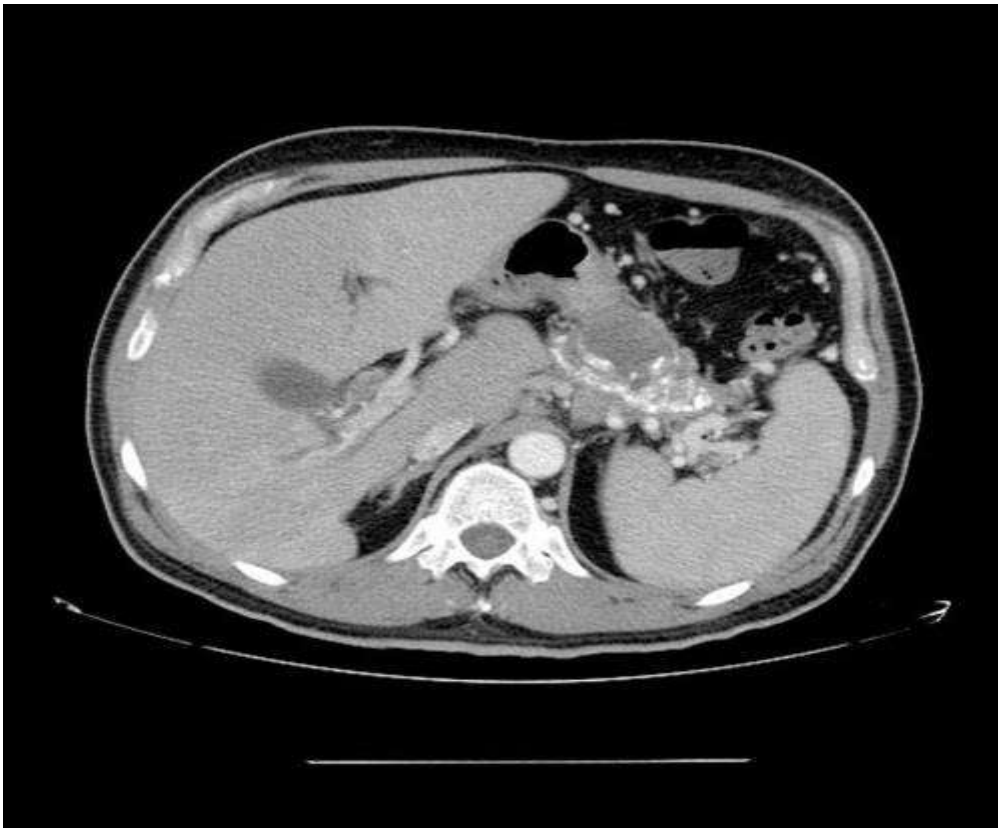
Management:

- pancreatic enzyme supplements
- analgesia
- antioxidants: limited evidence base - one study suggests benefit in early disease



© Image used on license from [Radiopaedia](#)

Multiple small calcific foci projected in the pancreas consistent with chronic pancreatitis



© Image used on license from [Radiopaedia](#)

CT showing an irregular shaped pancreas with the typical calcification of chronic pancreatitis

Clostridium difficile

Clostridium difficile is a Gram positive rod often encountered in hospital practice. It produces an exotoxin which causes intestinal damage leading to a syndrome called pseudomembranous colitis. *Clostridium difficile* develops when the normal gut flora are suppressed by broad-spectrum antibiotics. Clindamycin is historically associated with causing *Clostridium difficile* but the aetiology has evolved significantly over the past 10 years. Second and third generation cephalosporins are now the leading cause of *Clostridium difficile*.

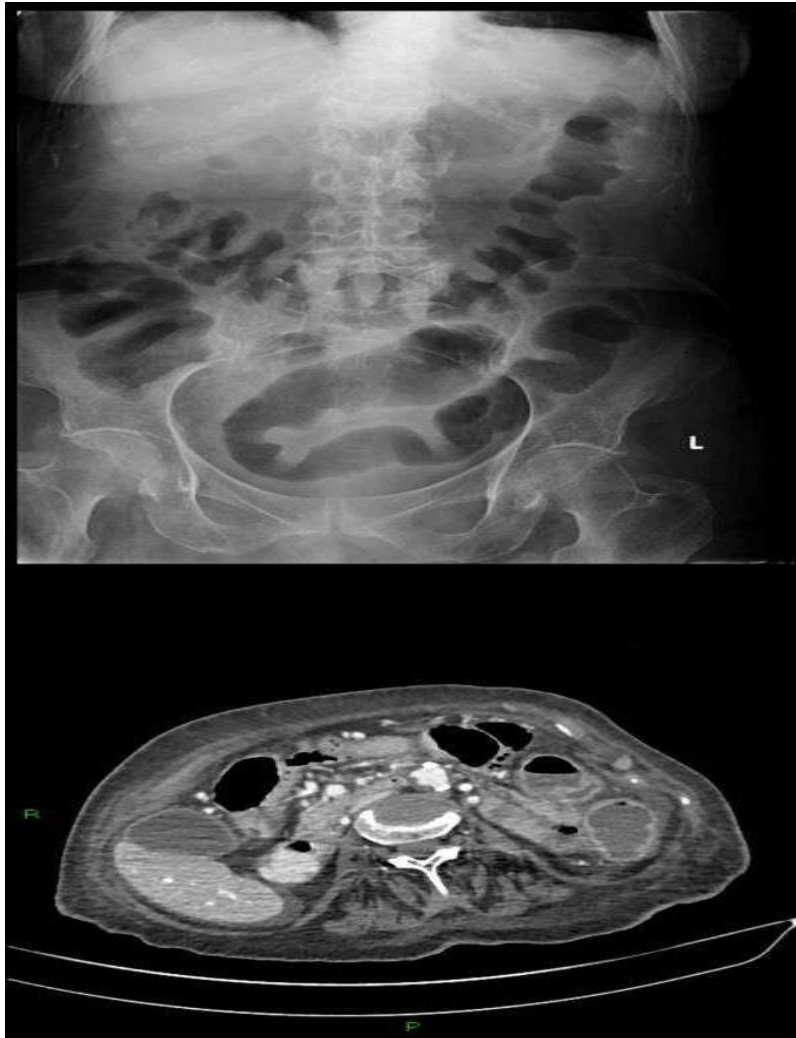
Features:

- diarrhoea
- abdominal pain
- a **raised white blood cell count** is characteristic
- if severe toxic megacolon may develop.

Diagnosis is made by detecting *Clostridium difficile* toxin (CDT) in the stool

Management:

- first-line therapy is oral metronidazole for 10-14 days
- if severe or not responding to metronidazole then oral vancomycin may be used
- for life-threatening infections a combination of oral vancomycin and intravenous metronidazole should be used.



© Image used on license from [Radiopaedia](http://radiopaedia.org)

Elderly lady with infectious colitis secondary to *Clostridium difficile*. On the abdominal film note the loss of bowel wall architecture and thumb-printing consistent with colitis. The CT from the same patient is enhanced by oral contrast. There is moderate free fluid in pelvis and peritoneum. The colon is oedematous throughout with enhancing walls, but of normal calibre. The sigmoid colon is smooth and featureless. Small bowel, liver, spleen, kidneys, adrenals and pancreas are normal.

External Links:

[Health Protection Agency](#)

2013 Clostridium difficile guideline

[Canadian Medical Journal](#)

Relative risk of developing Clostridium difficile

[NICE](#)

Clostridium difficile infection: risk with broad-spectrum antibiotic

[Royal College of Physicians](#)

Inpatient diarrhoea and *Clostridium difficile* infection

Coeliac disease

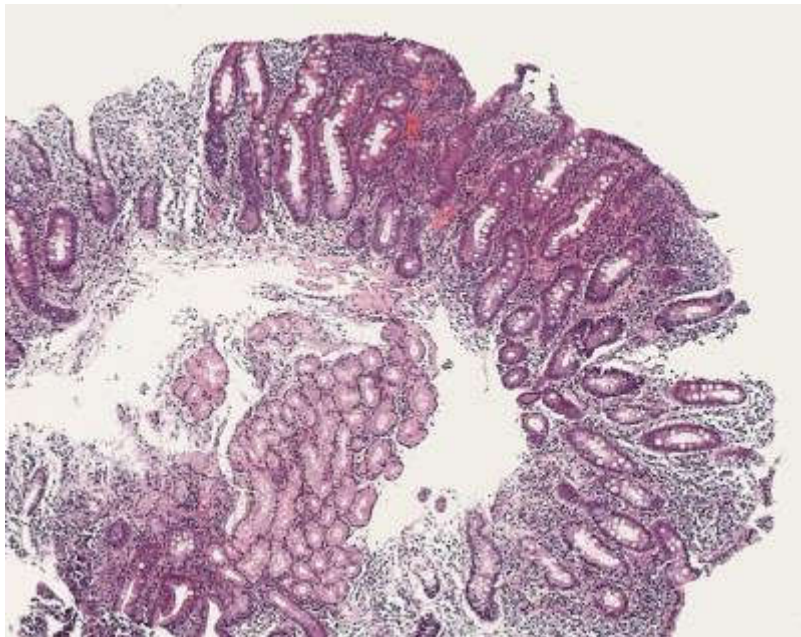
♦ Coeliac disease is caused by sensitivity to the protein gluten. Repeated exposure leads to villous atrophy which in turn causes malabsorption. Conditions associated with coeliac disease include dermatitis herpetiformis (a vesicular, pruritic skin eruption) and autoimmune disorders (type 1 diabetes mellitus and autoimmune hepatitis). It is strongly associated with HLA-DQ2 (95% of patients) and HLA-B8 (80%) as well as HLA-DR3 and HLA-DR7

In 2009 NICE issued guidelines on the investigation of coeliac disease. **They suggest that the following patients should be screened for coeliac disease:**

Signs and symptoms	Conditions
• Chronic or intermittent diarrhoea • Failure to thrive or faltering growth (in children) • Persistent or unexplained gastrointestinal symptoms including nausea and vomiting • Prolonged fatigue ('tired all the time') • Recurrent abdominal pain, cramping or distension • Sudden or unexpected weight loss • Unexplained iron-deficiency anaemia, or other unspecified anaemia	• Autoimmune thyroid disease • Dermatitis herpetiformis • Irritable bowel syndrome • Type 1 diabetes • First-degree relatives (parents, siblings or children) with coeliac disease

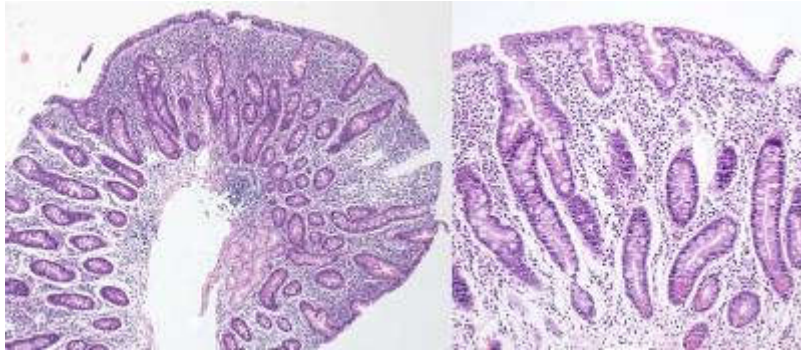
Complications

- anaemia: iron, folate and vitamin B12 deficiency (folate deficiency is more common than vitamin B12 deficiency in coeliac disease)
- hyposplenism
- osteoporosis, osteomalacia
- lactose intolerance
- enteropathy-associated T-cell lymphoma of small intestine
- subfertility, unfavourable pregnancy outcomes
- rare: oesophageal cancer, other malignancies



© Image used on license from PathoPic

Duodenal biopsy from a patient with coeliac disease. Complete atrophy of the villi with flat mucosa and marked crypt hyperplasia. Intraepithelial lymphocytosis. Dense mixed inflammatory infiltrate in the lamina propria.



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Duodenal biopsy from a patient with coeliac disease. Flat mucosa with hyperplastic crypts and dense cellular infiltrate in the lamina propria. Increased number of intraepithelial lymphocytes and vacuolated superficial epithelial cells. Higher magnification image on the right.

External Links:

[NICE](#)

2015 Coeliac disease guidelines

[British Society of Gastroenterology](#)

2002 coeliac disease guidelines

Coeliac disease: investigation

♦ Coeliac disease is caused by sensitivity to the protein gluten. Repeated exposure leads to villous atrophy which in turn causes malabsorption. Conditions associated with coeliac disease include dermatitis herpetiformis (a vesicular, pruritic skin eruption) and autoimmune disorders (type 1 diabetes mellitus and autoimmune hepatitis).

♦ Diagnosis is made by a combination of immunology and jejunal biopsy. Villous atrophy and immunology normally reverses on a gluten-free diet.

♦ NICE issued guidelines on the investigation of coeliac disease in 2009. If patients are already taking a gluten-free diet they should be asked, if possible, to reintroduce gluten for at least 6 weeks prior to testing.

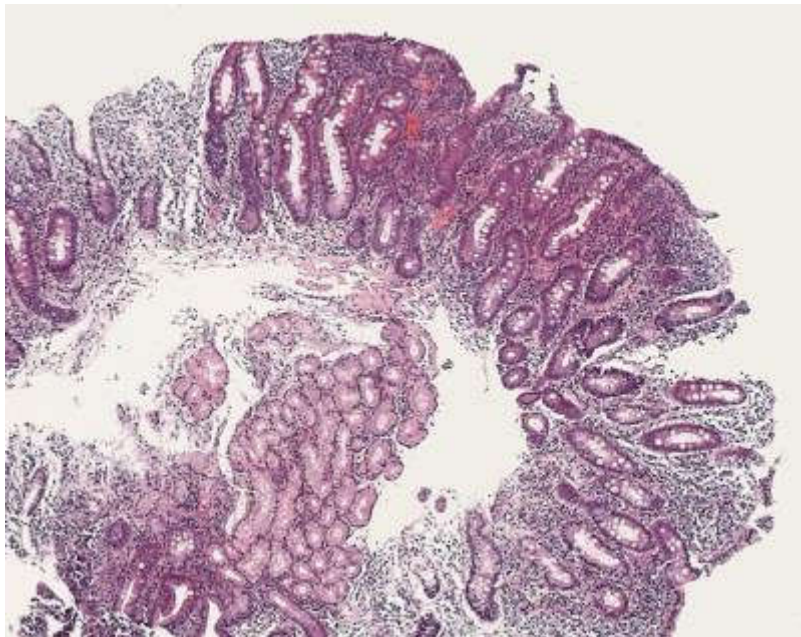
Immunology:

- tissue transglutaminase (TTG) antibodies (IgA) are first-choice according to NICE
- endomyseal antibody (IgA)
- anti-gliadin antibody (IgA or IgG) tests are not recommended by NICE
- anti-casein antibodies are also found in some patients

Jejunal biopsy

- villous atrophy
- crypt hyperplasia
- increase in intraepithelial lymphocytes
- lamina propria infiltration with lymphocytes

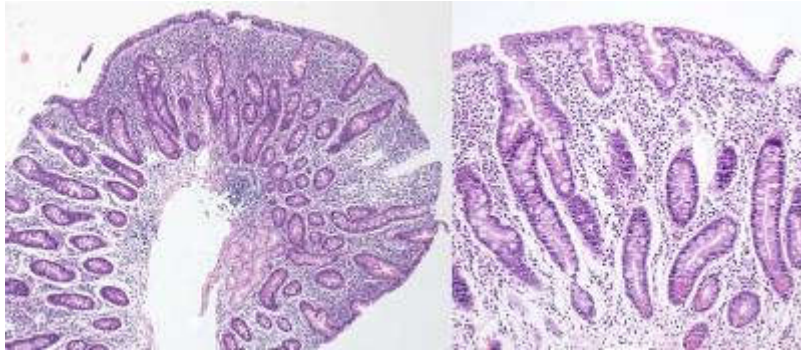
Rectal gluten challenge has been described but is not widely used



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Duodenal biopsy from a patient with coeliac disease. Complete atrophy of the villi with flat mucosa and marked crypt hyperplasia. Intraepithelial lymphocytosis. Dense mixed inflammatory infiltrate in the lamina propria.



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Duodenal biopsy from a patient with coeliac disease. Flat mucosa with hyperplastic crypts and dense cellular infiltrate in the lamina propria. Increased number of intraepithelial lymphocytes and vacuolated superficial epithelial cells. Higher magnification image on the right.

External Links:

[NICE](#)

2015 coeliac disease guidelines

[British Society of Gastroenterology](#)

2002 coeliac disease guidelines

Coeliac disease: management

The management of coeliac disease involves a **gluten-free diet**. Gluten containing cereals include:

- wheat: bread, pasta, pastry
- barley*: beer
- rye
- oats**

Some notable foods which are gluten-free include:

- rice
- potatoes
- corn (maize).

Tissue transglutaminase antibodies may be checked to check compliance with a gluten free diet.

♦Patients with coeliac disease often have a degree of **functional hyposplenism**. For this reason all patients with coeliac disease are offered the pneumococcal vaccine. Current guidelines suggest giving the influenza vaccine on an individual basis.

*whisky is made using malted barley. Proteins such as gluten are however removed during the distillation process making it safe to drink for patients with coeliac disease

**some patients with coeliac disease appear able to tolerate oats

External Links:

[NICE](#)

2015 coeliac disease guidelines

Colorectal cancer: genetics

It is currently thought there are three types of colon cancer:

- sporadic (95%)
- hereditary non-polyposis colorectal carcinoma (HNPCC, 5%)
- familial adenomatous polyposis (FAP, <1%)

Studies have shown that sporadic colon cancer may be due to a series of genetic mutations. For example, more than half of colon cancers show allelic loss of the APC gene. It is believed a further series of gene abnormalities e.g. activation of the K-ras oncogene, deletion of p53 and DCC tumour suppressor genes lead to invasive carcinoma

HNPCC, an autosomal dominant condition, is the most common form of inherited colon cancer. Around 90% of patients develop cancers, often of the proximal colon, which are usually poorly differentiated and highly aggressive. Currently seven mutations have been identified, which affect genes involved in DNA mismatch repair leading to microsatellite instability. **The most common genes involved are:**

- MSH2 (60% of cases)
- MLH1 (30%)

Patients with HNPCC are also at a higher risk of other cancers, with endometrial cancer being the next most common association, after colon cancer.

The Amsterdam criteria are sometimes used to aid diagnosis:

- at least 3 family members with colon cancer
- the cases span at least two generations
- at least one case diagnosed before the age of 50 years.

♦FAP is a rare autosomal dominant condition which leads to the formation of hundreds of polyps by the age of 30-40 years. Patients inevitably develop carcinoma. It is due to a mutation in a tumour suppressor gene called adenomatous polyposis coli gene (APC), located on chromosome 5. Genetic testing can be done by analysing DNA from a patient's white blood cells. Patients generally have a total colectomy with ileo-anal pouch formation in their twenties.

♦Patients with FAP are also at risk from duodenal tumours. A variant of FAP called Gardner's syndrome can also feature osteomas of the skull and mandible, retinal pigmentation, thyroid carcinoma and epidermoid cysts on the skin

External Links:

[SIGN](#)

Management of colorectal cancer guidelines

Crohn's disease

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus.

Pathology

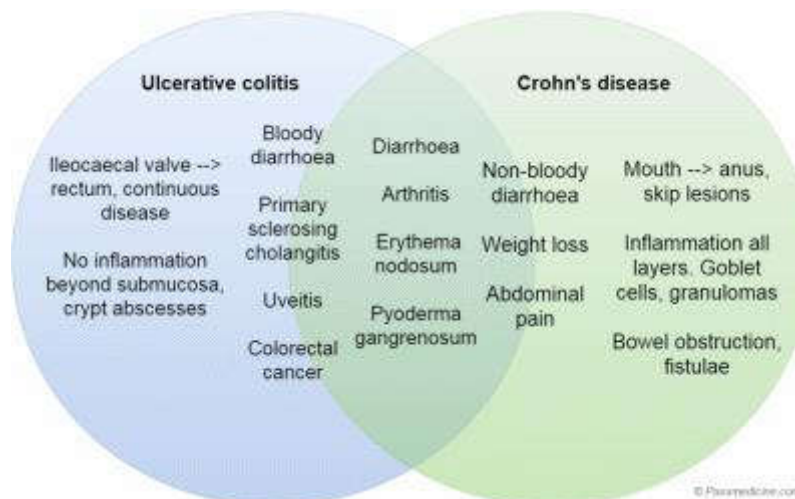
- cause is unknown but there is a strong genetic susceptibility
- inflammation occurs in all layers, down to the serosa. This is why patients with Crohn's are prone to strictures, fistulas and adhesions

Crohn's disease typically presents in late adolescence or early adulthood. **Features include:**

- presentation may be non-specific symptoms such as weight loss and lethargy
- diarrhoea: the most prominent symptom in adults. Crohn's colitis may cause bloody diarrhoea
- abdominal pain: the most prominent symptom in children
- perianal disease: e.g. Skin tags or ulcers
- extra-intestinal features are more common in patients with colitis or perianal disease.

Questions regarding the 'extra-intestinal' features of inflammatory bowel disease are common:

	Common to both Crohn's disease (CD) and Ulcerative colitis (UC)	Notes
Related to disease activity	Arthritis: pauciarticular, asymmetric Erythema nodosum Episcleritis Osteoporosis	Arthritis is the most common extra-intestinal feature in both CD and UC Episcleritis is more common in CD
Unrelated to disease activity	Arthritis: polyarticular, symmetric Uveitis Pyoderma gangrenosum Clubbing Primary sclerosing cholangitis	Primary sclerosing cholangitis is much more common in UC Uveitis is more common in UC



Venn diagram showing shared features and differences between ulcerative colitis and Crohn's disease. Note that whilst some features are present in both, some are much more common in one of the conditions, for example colorectal cancer in ulcerative colitis

External Links:

[NICE](#)

2012 Crohn disease guidelines

Crohn's disease: investigation

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus

Bloods

- C-reactive protein correlates well with disease activity

Endoscopy

- colonoscopy is the investigation of choice
- features suggest of Crohn's include deep ulcers, skip lesions

Histology

- inflammation in all layers from mucosa to serosa
- goblet cells
- granulomas

Small bowel enema

- high sensitivity and specificity for examination of the terminal ileum
- strictures: 'Kantor's string sign'
- proximal bowel dilation
- 'rose thorn' ulcers
- fistulae



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Barium study is shown from a patient with worsening Crohn's disease. Long segment of narrowed terminal ileum in a 'string like' configuration in keeping with a long stricture segment. Termed 'Kantor's string sign'.

Crohn's disease: management

Crohn's disease is a form of inflammatory bowel disease. It commonly affects the terminal ileum and colon but may be seen anywhere from the mouth to anus. NICE published guidelines on the management of Crohn's disease in 2012.

General points

- patients should be strongly advised to stop smoking
- some studies suggest an increased risk of relapse secondary to NSAIDs and the combined oral contraceptive pill but the evidence is patchy.

Inducing remission

- glucocorticoids (oral, topical or intravenous) are generally used to induce remission. Budesonide is an alternative in a subgroup of patients
- enteral feeding with an elemental diet may be used in addition to or instead of other measures to induce remission, particularly if there is concern regarding the side-effects of steroids (for example in young children).
- 5-ASA drugs (e.g. mesalazine) are used second-line to glucocorticoids but are not as effective.

Chapter: Gastroenterology

- azathioprine or mercaptopurine* may be used as an add-on medication to induce remission but is not used as monotherapy. Methotrexate is an alternative to azathioprine
- infliximab is useful in refractory disease and fistulating Crohn's. Patients typically continue on azathioprine or methotrexate
- metronidazole is often used for isolated peri-anal disease

Maintaining remission

- as above, stopping smoking is a priority (remember: smoking makes Crohn's worse, but may help ulcerative colitis)
- azathioprine or mercaptopurine is used first-line to maintain remission
- methotrexate is used second-line
- 5-ASA drugs (e.g. mesalazine) should be considered if a patient has had previous surgery

Surgery

- around 80% of patients with Crohn's disease will eventually have surgery
- see below for further detail

Surgical interventions in Crohn's disease

♦The commonest disease pattern in Crohn's is stricturing terminal ileal disease and this often culminates in an **ileocaecal resection**. Other procedures performed include segmental small bowel resections and stricturoplasty. Colonic involvement in patients with Crohn's is not common and, where found, distribution is often segmental. However, despite this distribution segmental resections of the colon in patients with Crohn's disease are generally not advocated because the recurrence rate in the remaining colon is extremely high, as a result, the standard options of colonic surgery in Crohn's patients are generally; sub total colectomy, panproctocolectomy and staged sub total colectomy and proctectomy. Restorative procedures such as ileoanal pouch have no role in therapy.

♦Crohn's disease is notorious for the development of intestinal fistulae; these may form between the rectum and skin (perianal) or the small bowel and skin. Fistulation between loops of bowel may also occur and result in bacterial overgrowth and malabsorption. Management of enterocutaneous fistulae involves controlling sepsis, optimising nutrition, imaging the disease and planning definitive surgical management.

*assess thiopurine methyltransferase (TPMT) activity before offering azathioprine or mercaptopurine

External Links:

[NICE](#)

2012 Crohns guidelines

[British Society of Gastroenterology](#)

2004 IBD guidelines

[Clinical Knowledge Summaries](#)

Crohns disease guidelines

Diphtheria

Diphtheria is caused by the Gram positive bacterium *Corynebacterium diphtheriae*

Pathophysiology:

- releases an exotoxin encoded by a β -prophage
- exotoxin inhibits protein synthesis by catalyzing ADP-ribosylation of elongation factor EF-2

Diphtheria toxin commonly causes a 'diphtheric membrane' on tonsils caused by necrotic mucosal cells. Systemic distribution may produce necrosis of myocardial, neural and renal tissue

Possible presentations:

- recent visitors to Eastern Europe/Russia/Asia
- sore throat with a 'diphtheric membrane' - see above
- bulky cervical lymphadenopathy
- neuritis e.g. cranial nerves
- heart block

Diverticulosis

Diverticulosis is an extremely common disorder characterised by multiple outpouchings of the bowel wall, most commonly in the sigmoid colon. Strictly speaking the term diverticular disease is reserved for patients who are symptomatic - diverticulosis is the more accurate term for diverticula being present.

Diverticulosis can present in a number of ways:

- painful diverticular disease: altered bowel habit, colicky left sided abdominal pain. A high fibre diet is usually recommended to minimise symptoms
- diverticulitis: see below for more details

Diverticulitis

One of the diverticular become infected. The classical presentation is:

- left iliac fossa pain and tenderness
- anorexia, nausea and vomiting
- diarrhoea
- features of infection (pyrexia, raised WBC and CRP)

Management:

- mild attacks can be treated with oral antibiotics
- more significant episodes are managed in hospital. Patients are made nil by mouth, intravenous fluids and intravenous antibiotics (typical a cephalosporin + metronidazole) are given

Complications of diverticulitis include:

- abscess formation
- peritonitis
- obstruction
- perforation

Drug-induced liver disease

Drug-induced liver disease is generally divided into hepatocellular, cholestatic or mixed. There is however considerable overlap, with some drugs causing a range of changes to the liver

The following drugs tend to cause a hepatocellular picture:

- paracetamol
- sodium valproate, phenytoin
- MAOIs
- halothane
- anti-tuberculosis: isoniazid, rifampicin, pyrazinamide
- statins
- alcohol
- amiodarone
- methyldopa
- nitrofurantoin

The following drugs tend to cause cholestasis (+/- hepatitis):

- oral contraceptive pill
- antibiotics: flucloxacillin, co-amoxiclav, erythromycin*
- anabolic steroids, testosterone
- phenothiazines: chlorpromazine, prochlorperazine
- sulphonylureas
- fibrates
- rare reported causes: nifedipine

Liver cirrhosis:

- methotrexate
- methyldopa
- amiodarone

*risk may be reduced with erythromycin stearate

External Links:

[LiverTox](#)

Database of drugs causing liver injury

Drugs causing dyspepsia

Causes

- NSAIDs
- bisphosphonates
- steroids

The following drugs may cause reflux by reducing lower oesophageal sphincter (LOS) pressure:

- calcium channel blockers*
- nitrates*
- theophyllines

*calcium channel blockers and nitrates are occasionally used in the management of achalasia, itself a cause of dyspepsia, because of their effect on the LOS.

Dubin-Johnson syndrome

Dubin-Johnson syndrome is a benign autosomal recessive disorder resulting in hyperbilirubinaemia (conjugated, therefore present in urine). It is due to a defect in the canalicular multispecific organic anion transporter (cMOAT) protein. This causes defective hepatic bilirubin excretion

Dyspepsia

The 2015 NICE guidelines 'Suspected cancer: recognition and referral' further updated the advice on who needs urgent referral for an endoscopy (i.e. within 2 weeks). The list below combines the advice for oesophageal and stomach cancer, with the bold added by the author, not NICE.

Urgent

All patient who've got **dysphagia**

All patient who've got an **upper abdominal mass** consistent with stomach cancer

Patients aged ≥ 55 years who've got **weight loss**, **AND any of the following**:

- upper abdominal pain
- reflux
- dyspepsia

Non-urgent

Patients with **haematemesis**

Patients aged ≥ 55 years who've got:

- **treatment-resistant dyspepsia** or
- upper abdominal pain with low haemoglobin levels or
- **raised platelet count** with any of the following: nausea, vomiting, weight loss, reflux, dyspepsia, upper abdominal pain
- nausea or vomiting with any of the following: weight loss, reflux, dyspepsia, upper abdominal pain

Managing patients who do not meet referral criteria ('undiagnosed dyspepsia')

This can be summarised at a step-wise approach:

1. Review medications for possible causes of dyspepsia
2. Lifestyle advice
3. Trial of full-dose proton pump inhibitor for one month OR a 'test and treat' approach for *H. pylori*.

Chapter: Gastroenterology

Testing for *H. pylori* infection

- initial diagnosis: NICE recommend using a carbon-13 urea breath test or a stool antigen test, or laboratory-based serology 'where its performance has been locally validated'
- test of cure: carbon-13 urea breath test

External Links:

[NICE](#)

2014 dyspepsia guideline

[NICE](#)

2015 Suspected cancer: recognition and referral

Dysphagia

The table below gives characteristic exam question features for conditions causing dysphagia:

Causes	Notes
Oesophageal cancer	Dysphagia may be associated with weight loss, anorexia or vomiting during eating Past history may include Barrett's oesophagus, GORD, excessive smoking or alcohol use
Oesophagitis	May be history of heartburn Odynophagia but no weight loss and systemically well
Oesophageal candidiasis	There may be a history of HIV or other risk factors such as steroid inhaler use
Achalasia	Dysphagia of both liquids and solids from the start Heartburn Regurgitation of food - may lead to cough, aspiration pneumonia etc
Pharyngeal pouch	More common in older men Represents a posteromedial herniation between thyropharyngeus and cricopharyngeus muscles Usually not seen but if large then a midline lump in the neck that gurgles on palpation Typical symptoms are dysphagia, regurgitation, aspiration and chronic cough. Halitosis may occasionally be seen

Chapter: Gastroenterology

Systemic sclerosis	Other features of CREST syndrome may be present, namely Calcinosis, Raynaud's phenomenon, oesophageal dysmotility, Sclerodactyly, Telangiectasia As well as oesophageal dysmotility the lower oesophageal sphincter (LES) pressure is decreased. This contrasts to achalasia where the LES pressure is increased
Myasthenia gravis	Other symptoms may include extraocular muscle weakness or ptosis Dysphagia with liquids as well as solids
Globus hystericus	May be history of anxiety Symptoms are often intermittent and relieved by swallowing Usually painless - the presence of pain should warrant further investigation for organic causes

Causes of dysphagia - by classification

As with many conditions, it's often useful to think about causes of a symptom in a structured way:

Classification	Examples
Extrinsic	• Mediastinal masses • Cervical spondylosis
Oesophageal wall	• Achalasia • Diffuse oesophageal spasm • Hypertensive lower oesophageal sphincter
Intrinsic	• Tumours • Strictures • Oesophageal web • Schatzki rings
Neurological	• CVA • Parkinson's disease • Multiple Sclerosis • Brainstem pathology • Myasthenia Gravis

Investigation:

♦ All patients require an upper GI endoscopy unless there are compelling reasons for this not to be performed. Motility disorders may be best appreciated by undertaking fluoroscopic swallowing studies.

♦ A full blood count should be performed.

♦ Ambulatory oesophageal pH and manometry studies will be required to evaluate conditions such as achalasia and patients with GORD being considered for fundoplication surgery.

Exotoxins and endotoxins

Exotoxins are secreted by bacteria where as endotoxins are only released following lysis of the cell. Exotoxins are generally released by Gram positive bacteria with the notable exceptions of *Vibrio cholerae* and some strains of *E. coli*

It is possible to classify exotoxins by their primary effects:

- pyrogenic toxins
- enterotoxins
- neurotoxins
- tissue invasive toxins
- miscellaneous toxins

Pyrogenic toxins

Pyrogenic toxins stimulate the release of endogenous cytokines resulting in fever, rash etc. They are superantigens which bridge the MHC class II protein on antigen-presenting cells with the T cell receptor on the surface of T cells resulting in massive cytokine release.

Organism	Toxin	Notes
Staphylococcus aureus	Toxic shock syndrome (TSST-1 superantigen) toxin	Results in high fever, hypotension, exfoliative rash
Streptococcus pyogenes	Streptococcal pyrogenic exotoxin A & C	Results in scarlet fever

Enterotoxins:

Enterotoxins act on the gastrointestinal tract causing one of two patterns of illness:

- diarrhoeal illness
- vomiting illness ('food poisoning')

Organism	Toxin	Notes
<i>Vibrio cholerae</i>	Cholera toxin	Causes activation of adenylate cyclase (via G _s) leading to increases in cAMP levels, which in turn leads to increased chloride secretion and reduced sodium absorption
<i>Shigella dysenteriae</i>	Shiga toxin	Inactivates 60S ribosome → epithelial cell death
<i>Escherichia coli</i>	1. Heat labile toxin 2. Heat stable toxin	1. Activates adenylate cyclase (via G _s), increasing cAMP → watery diarrhoea 2. Activates guanylate cyclase, increasing cGMP → watery diarrhoea
<i>Staphylococcus aureus</i>	<i>Staphylococcus aureus</i> enterotoxin	Vomiting and diarrhoeal illness lasting < 24 hours
<i>Bacillus cereus</i>	Cereulide	Potent cytotoxin that destroys mitochondria. Causes a vomiting illness which may present within 4 hours of ingestion

Neurotoxins

Neurotoxins act on the nerves (tetanus) or the neuromuscular junction (botulism) causing paralysis.

Organism	Toxin	Notes
<i>Clostridium tetani</i>	Tetanospasmin	Blocks the release of the inhibitory neurotransmitters GABA and glycine resulting in continuous motor neuron activity → continuous muscle contraction → lockjaw and respiratory paralysis
<i>Clostridium botulinum</i>	Botulinum toxin	Blocks acetylcholine (ACh) release leading to flaccid paralysis

Tissue invasive toxins

Organism	Toxin	Notes
<i>Clostridium perfringens</i>	α-toxin, a lecithinase	Causes gas gangrene (myonecrosis) and haemolysis
<i>Staphylococcus aureus</i>	Exfoliatin	Staphylococcal scalded skin syndrome

Miscellaneous toxins

Organism	Toxin	Notes
Corynebacterium diphtheriae	Diphtheria toxin	ADP ribosylates elongation factor (EF-2), resulting in inhibition, causing a 'diphtheric membrane' on tonsils caused by necrotic mucosal cells. Systemic distribution may produce necrosis of myocardial, neural and renal tissue
Pseudomonas aeruginosa	Exotoxin A	Also inhibits EF-2 by the same mechanism as above
Bacillus anthracis	Oedema factor (EF)	Forms a calmodulin-dependent adenylate cyclase which increases cAMP, impairing the function of neutrophils/macrophages → reduced phagocytosis
Bordetella pertussis	Pertussis exotoxin	Inhibits G _i leading to increases in cAMP levels, impairing the function of neutrophils/macrophages → reduced phagocytosis

Endotoxins

Endotoxins are lipopolysaccharides that are released from Gram-negative bacteria such as *Neisseria meningitidis*.

External Links:

[Postgraduate Medical Journal](#)

Mechanisms of bacterial pathogenicity

Gallstones

◆Up to 24% of women and 12% of men may have gallstones. Of these up to 30% may develop local infection and cholecystitis. In patients subjected to surgery, 12% will have stones contained within the common bile duct. The majority of gallstones are of a mixed composition (50%) with pure cholesterol stones accounting for 20% of cases.

◆The aetiology of CBD stones differs in the world, in the West most CBD stones are the result of migration. In the East, a far higher proportion arise in the CBD de novo.

◆The classical symptoms are of colicky right upper quadrant pain that occurs postprandially. The symptoms are usually worst following a fatty meal when cholecystokin levels are highest and gallbladder contraction is maximal.

Investigation

In almost all suspected cases the standard diagnostic workup consists of abdominal ultrasound and liver function tests. Of patients who have stones within the bile duct, 60% will have at least one abnormal result on LFT's. Ultrasound is an important test, but is operator dependent and therefore may occasionally need to be repeated if a negative result is at odds with the clinical picture. Where stones are suspected in the bile duct the options lie between magnetic resonance cholangiography and intraoperative imaging. The choice between these two options is determined by the skills and experience of the surgeon. The advantages of intraoperative imaging are less useful in making therapeutic decisions if the operator is unhappy about proceeding the bile duct exploration, and in such circumstances, preoperative MRCP is probably a better option.

Specific gallstone and gallbladder related disease

Disease	Features	Management
Biliary colic	Colicky abdominal pain, worse postprandially, worse after fatty foods	If imaging shows gallstones and history compatible then laparoscopic cholecystectomy
Acute cholecystitis	Right upper quadrant pain Fever Murphys sign on examination Occasionally mildly deranged LFT's (especially if Mirizzi syndrome)	Imaging (USS) and cholecystectomy (ideally within 48 hours of presentation) (2)

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Gallbladder abscess	Usually prodromal illness and right upper quadrant pain Swinging pyrexia Patient may be systemically unwell Generalised peritonism not present	Imaging with USS +/- CT Scanning Ideally, surgery although subtotal cholecystectomy may be needed if Calot's triangle is hostile In unfit patients, percutaneous drainage may be considered
Cholangitis	Patient severely septic and unwell Jaundice Right upper quadrant pain	Fluid resuscitation Broad-spectrum intravenous antibiotics Correct any coagulopathy Early ERCP
Gallstone ileus	Patients may have a history of previous cholecystitis and known gallstones Small bowel obstruction (may be intermittent)	Laparotomy and removal of the gallstone from small bowel, the enterotomy must be made proximal to the site of obstruction and not at the site of obstruction. The fistula between the gallbladder and duodenum should not be interfered with.
Acalculous cholecystitis	Patients with intercurrent illness (e.g. diabetes, organ failure) Patient of systemically unwell Gallbladder inflammation in absence of stones High fever	If patient fit then cholecystectomy, if unfit then percutaneous cholecystostomy

Treatment

◆ Asymptomatic gallstones which are located in the gallbladder are common and do not require treatment. However, if stones are present in the common bile duct there is an increased risk of complications such as cholangitis or pancreatitis and surgical management should be considered.

◆ Patients with asymptomatic gallstones rarely develop symptoms related to them (less than 2% per year) and may, therefore, be managed expectantly. In almost all cases of symptomatic gallstones the treatment of choice is cholecystectomy performed via the laparoscopic route. In the very frail patient, there is sometimes a role for the selective use of ultrasound guided cholecystostomy.

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◆During the course of the procedure, some surgeons will routinely perform either intraoperative cholangiography to either confirm anatomy or to exclude CBD stones.

◆The latter may be more easily achieved by use of laparoscopic ultrasound. If stones are found then the options lie between early ERCP in the day or so following surgery or immediate surgical exploration of the bile duct. When performed via the trans cystic route this adds little in the way of morbidity and certainly results in faster recovery. Where transcystic exploration fails the alternative strategy is that of formal choledochotomy.

◆The exploration of a small duct is challenging and ducts of less than 8mm should not be explored.

◆Small stones that measure less than 5mm may be safely left and most will pass spontaneously.

Risks of ERCP(1):

- Bleeding 0.9% (rises to 1.5% if sphincterotomy performed)
- Duodenal perforation 0.4%
- Cholangitis 1.1%
- Pancreatitis 1.5%

References

1. Williams E *et al.* Guidelines on the management of common bile duct stones (CBDS)*Gut* 2008;57:10041021
2. Gurusamy KS, Samraj K. Early versus delayed laparoscopic cholecystectomy for acute cholecystitis. *Cochrane Database Syst Rev.* 2006 Oct 18;(4):CD005440.

Gastric MALT lymphoma

Overview

- associated with *H. pylori* infection in 95% of cases
- good prognosis
- if low grade then 80% respond to *H. pylori* eradication

Features

- paraproteinaemia may be present

External Links:

[Clinical Microbiology Reviews](#)

Helicobacter pylori and Gastric Cancer: Factors That Modulate Disease Risk

Gastrointestinal physiology: acid secretion

Principle mediators of acid secretion:

- gastrin
- vagal stimulation
- histamine

Factors increasing acid secretion:

- gastrinoma
- small bowel resection (removal of inhibition)
- systemic mastocytosis (elevated histamine levels)
- basophilia

Factors decreasing acid secretion:

- drugs: H2-antagonists, PPIs
- hormones: secretin, VIP, GIP, CCK

Gilbert's syndrome

Gilbert's syndrome is an autosomal recessive* condition of defective bilirubin conjugation due to a deficiency of UDP glucuronyl transferase. The prevalence is approximately 1-2% in the general population

Features

- unconjugated hyperbilinaemia (i.e. not in urine)
- jaundice may only be seen during an intercurrent illness

Investigation and management:

- investigation: rise in bilirubin following prolonged fasting or IV nicotinic acid
- no treatment required

*the exact mode of inheritance is still a matter of debate

GORD: investigation

Overview

- poor correlation between symptoms and endoscopy appearance

Indications for upper GI endoscopy:

- age > 55 years
- symptoms > 4 weeks or persistent symptoms despite treatment
- dysphagia
- relapsing symptoms
- weight loss

If endoscopy is negative consider 24-hr oesophageal pH monitoring (the gold standard test for diagnosis)

Haemochromatosis: features

Haemochromatosis is an autosomal recessive disorder of iron absorption and metabolism resulting in iron accumulation. It is caused by inheritance of mutations in the HFE gene on both copies of chromosome 6*. It is often asymptomatic in early disease and initial symptoms often non-specific e.g. lethargy and arthralgia

Epidemiology

- 1 in 10 people of European descent carry a mutation genes affecting iron metabolism, mainly HFE
- prevalence in people of European descent = 1 in 200

Presenting features:

- early symptoms include fatigue, erectile dysfunction and arthralgia (often of the hands)
- 'bronze' skin pigmentation
- diabetes mellitus
- liver: stigmata of chronic liver disease, hepatomegaly, cirrhosis, hepatocellular deposition)
- cardiac failure (2nd to dilated cardiomyopathy)
- hypogonadism (2nd to cirrhosis and pituitary dysfunction - hypogonadotrophic hypogonadism)
- arthritis (especially of the hands)

Questions have previously been asked regarding which features are reversible with treatment:

Reversible complications	Irreversible complications
• Cardiomyopathy • Skin pigmentation	• Liver cirrhosis** • Diabetes mellitus • Hypogonadotrophic hypogonadism • Arthropathy

*there are rare cases of families with classic features of genetic haemochromatosis but no mutation in the HFE gene

**whilst elevated liver function tests and hepatomegaly may be reversible, cirrhosis is not

Haemochromatosis: investigation

Haemochromatosis is an autosomal recessive disorder of iron absorption and metabolism resulting in iron accumulation. It is caused by inheritance of mutations in the HFE gene on both copies of chromosome 6*.

There is continued debate about the best investigation to screen for haemochromatosis.

- general population: transferrin saturation is considered the most useful marker. Ferritin should also be measured but is not usually abnormal in the early stages of iron accumulation
- testing family members: genetic testing for HFE mutation

These guidelines may change as HFE gene analysis become less expensive

Diagnostic tests

- molecular genetic testing for the C282Y and H63D mutations
- liver biopsy: Perl's stain

Typical iron study profile in patient with haemochromatosis:

- transferrin saturation > 55% in men or > 50% in women
- raised ferritin (e.g. > 500 ug/l) and iron
- low TIBC.

Monitoring adequacy of venesection:

- transferrin saturation should be kept below 50% and the serum ferritin concentration below 50 ug/l

Joint x-rays characteristically show chondrocalcinosis

*there are rare cases of families with classic features of genetic haemochromatosis but no mutation in the HFE gene

External Links:

[European Association for the Study of the Liver](#)

2010 Haemochromatosis guidelines

Helicobacter pylori

Helicobacter pylori is a Gram negative bacteria associated with a variety of gastrointestinal problems, principally peptic ulcer disease

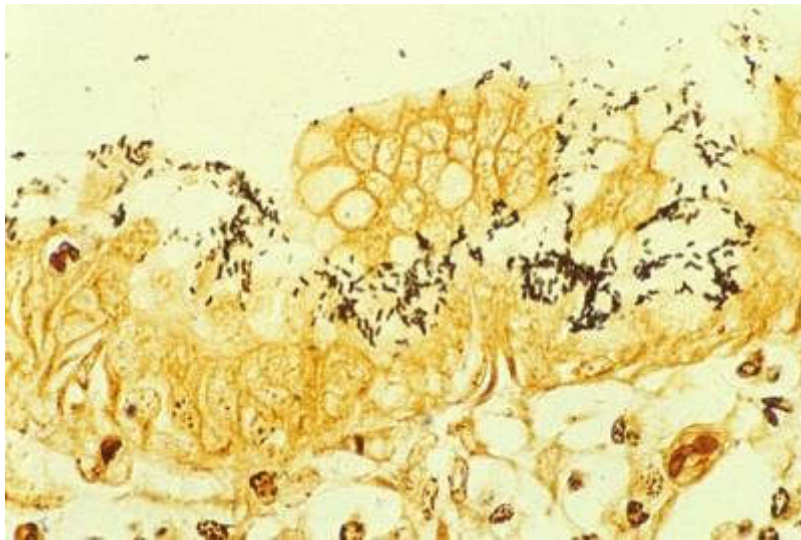
Associations:

- peptic ulcer disease (95% of duodenal ulcers, 75% of gastric ulcers)
- gastric cancer
- B cell lymphoma of MALT tissue (eradication of H pylori results causes regression in 80% of patients)
- atrophic gastritis

The role of H pylori in Gastro-oesophageal reflux disease (GORD) is unclear - there is currently no role in GORD for the eradication of H pylori

Management: - eradication may be achieved with a 7 day course of

- a proton pump inhibitor + amoxicillin + clarithromycin, or
- a proton pump inhibitor + metronidazole + clarithromycin



***H. pylori* colonized on the surface of regenerative epithelium (silver stain)**

External Links:

[NICE](#)

2014 Dyspepsia guidelines: *Helicobacter pylori* infection.

***Helicobacter pylori*: tests**

Urea breath test

- patients consume a drink containing carbon isotope 13 (¹³C) enriched urea
- urea is broken down by *H. pylori* urease
- after 30 mins patient exhale into a glass tube
- mass spectrometry analysis calculates the amount of ¹³C CO₂
- should not be performed within 4 weeks of treatment with an antibacterial or within 2 weeks of an antisecretory drug (e.g. a proton pump inhibitor)
- sensitivity 95-98%, specificity 97-98%

Rapid urease test (e.g. CLO test):

- biopsy sample is mixed with urea and pH indicator
- colour change if *H. pylori* urease activity
- sensitivity 90-95%, specificity 95-98%

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Serum antibody:

- remains positive after eradication
- sensitivity 85%, specificity 80%

Culture of gastric biopsy

- provide information on antibiotic sensitivity
- sensitivity 70%, specificity 100%

Gastric biopsy:

- histological evaluation alone, no culture
- sensitivity 95-99%, specificity 95-99%

Stool antigen test:

- sensitivity 90%, specificity 95%

External Links:

[Public Health England](#)

Helicobacter Pylori: Diagnosis and Treatment

Hepatitis C

Hepatitis C is likely to become a significant public health problem in the UK in the next decade. It is thought around 200,000 people are chronically infected with the virus. At risk groups include intravenous drug users and patients who received a blood transfusion prior to 1991 (e.g. haemophiliacs).

Pathophysiology:

- hepatitis C is a RNA flavivirus
- incubation period: 6-9 weeks

Transmission:

- the risk of transmission during a needle stick injury is about 2%
- the vertical transmission rate from mother to child is about 6%. The risk is higher if there is coexistent HIV
- breast feeding is not contraindicated in mothers with hepatitis C
- the risk of transmitting the virus during sexual intercourse is probably less than 5%

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Features

- after exposure to the hepatitis C virus less than 20% of patients develop an acute hepatitis

Complications:

- chronic infection (80-85%) - only 15-20% of patients will clear the virus after an acute infection and hence the majority will develop chronic hepatitis C
- cirrhosis (20-30% of those with chronic disease)
- hepatocellular cancer
- cryoglobulinaemia
- porphyria cutanea tarda (PCT): it is increasingly recognised that PCT may develop in patients with hepatitis C, especially if there are other factors such as alcohol abuse

Management of chronic infection:

- treatment depends on genotype
- currently a combination of pegylated interferon-alpha, ribavirin and a protease inhibitor (e.g. boceprevir, simeprevir and telaprevir) is used
- cure rates are now approaching 90%, including for some strains which have been previously difficult to treat
- the aim of treatment is sustained virological response (SVR), defined as undetectable

serum HCV RNA six months after the end of therapy

Complications of treatment:

- ribavirin - side-effects: haemolytic anaemia, cough. Women should not become pregnant within 6 months of stopping ribavirin as it is teratogenic
- interferon alpha - side-effects: flu-like symptoms, depression, fatigue, leukopenia, thrombocytopenia

External Links:

[SIGN](#)

2013 Management of hepatitis C

[Royal College of Physicians of London and the British Society of Gastroenterology](#)

Clinical Guidelines on the management of hepatitis C

Hepatitis D

♦Hepatitis D is a single stranded RNA virus that is transmitted parenterally. It is an incomplete RNA virus that requires hepatitis B surface antigen to complete its replication and transmission cycle.

♦It is transmitted in a similar fashion to hepatitis B (exchange of bodily fluids) and patients may be infected with hepatitis B and hepatitis D at the same time.

Hepatitis D terminology:

- Co-infection: Hepatitis B and Hepatitis D infection at the same time.
- Superinfection: A hepatitis B surface antigen positive patient subsequently develops a hepatitis D infection.

Superinfection is associated with high risk of fulminant hepatitis, chronic hepatitis status and cirrhosis.

Diagnosis is made via reverse polymerase chain reaction of hepatitis D RNA. Interferon is currently used as treatment, but with a poor evidence base.

Hepatobiliary disease and related disorders

The table below gives characteristic exam question features for conditions causing hepatobiliary disease and related disorders:

Condition	Features
Viral hepatitis	Common symptoms include: • nausea and vomiting, anorexia • myalgia • lethargy • right upper quadrant (RUQ) pain Questions may point to risk factors such as foreign travel or intravenous drug use.
Congestive hepatomegaly	The liver only usually causes pain if stretched. One common way this can occur is as a consequence of congestive heart failure. In severe cases cirrhosis may occur.
Acute cholecystitis	Pain similar to biliary colic but more severe and persistent. The pain may radiate to the back or right shoulder. The patient may be pyrexial and Murphy's sign positive (arrest of inspiration on palpation of the RUQ)

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Ascending cholangitis	An infection of the bile ducts commonly secondary to gallstones. Classically presents with a triad of: • fever (rigors are common) • RUQ pain • jaundice
Gallstone ileus	This describes small bowel obstruction secondary to an impacted gallstone. It may develop if a fistula forms between a gangrenous gallbladder and the duodenum. Abdominal pain, distension and vomiting are seen.
Cholangiocarcinoma	Persistent biliary colic symptoms, associated with anorexia, jaundice and weight loss. A palpable mass in the right upper quadrant (Courvoisier sign), periumbilical lymphadenopathy (Sister Mary Joseph nodes) and left supraclavicular adenopathy (Virchow node) may be seen
Acute pancreatitis	Usually due to alcohol or gallstones Severe epigastric pain Vomiting is common Examination may reveal tenderness, ileus and low-grade fever Periumbilical discoloration (Cullen's sign) and flank discoloration (Grey-Turner's sign) is described but rare
Pancreatic cancer	Painless jaundice is the classical presentation of pancreatic cancer. However pain is actually a relatively common presenting symptom of pancreatic cancer. Anorexia and weight loss are common
Amoebic liver abscess	Typical symptoms are malaise, anorexia and weight loss. The associated RUQ pain tends to be mild and jaundice is uncommon.

External Links:

[Gastrointestinal tract \(upper\) cancers - recognition and referral](#)

Hepatobiliary disease and related disorders

Hepatomegaly

Common causes of hepatomegaly

- **Cirrhosis:** if early disease, later liver decreases in size. Associated with a non-tender, firm liver
- **Malignancy:** metastatic spread or primary hepatoma. Associated with a hard, irregular. liver edge
- **Right heart failure:** firm, smooth, tender liver edge. May be pulsatile

Other causes

- viral hepatitis
- glandular fever
- malaria
- abscess: pyogenic, amoebic
- hydatid disease
- haematological malignancies
- haemochromatosis
- primary biliary cirrhosis
- sarcoidosis, amyloidosis

Hepatorenal syndrome: management

♦The management of hepatorenal syndrome (HRS) is notoriously difficult. The ideal treatment is liver transplantation but patients are often too unwell to have surgery and there is a shortage of donors

♦The most accepted theory regarding the pathophysiology of HRS is that vasoactive mediators cause **splanchnic vasodilation** which in turn reduces the systemic vascular resistance. This results in 'underfilling' of the kidneys. This is sensed by the juxtaglomerular apparatus which then activates the renin-angiotensin-aldosterone system, causing renal vasoconstriction which is not enough to counterbalance the effects of the splanchnic vasodilation.

Hepatorenal syndrome has been categorized into two types:

Type 1 HRS	Type 2 HRS
•Rapidly progressive •Doubling of serum creatinine to $> 221 \mu\text{mol/L}$ or a halving of the creatinine clearance to less than 20 ml/min over a period of less than 2 weeks •Very poor prognosis	•Slowly progressive •Prognosis poor, but patients may live for longer

Management options

- vasopressin analogues, for example terlipressin, have a growing evidence base supporting their use. They work by causing vasoconstriction of the splanchnic circulation
- volume expansion with 20% albumin
- transjugular intrahepatic portosystemic shunt

External Links:

[Patient.info](#)

Hepatorenal syndrome

Hepatosplenomegaly

Causes of hepatosplenomegaly

- chronic liver disease* with portal hypertension
- infections: glandular fever, malaria, hepatitis
- lymphoproliferative disorders
- myeloproliferative disorders e.g. CML
- amyloidosis

*the latter stages of cirrhosis are associated with a small liver

Hirschsprung's disease

Hirschsprung's disease is caused by an aganglionic segment of bowel due to a developmental failure of the parasympathetic Auerbach and Meissner plexuses. Although rare (occurring in 1 in 5,000 births) it is an important differential diagnosis in childhood constipation

Possible presentations

- neonatal period e.g. failure or delay to pass meconium
- older children: constipation, abdominal distension

Associations

- 3 times more common in males
- Down's syndrome

HIV: biliary and pancreatic disease

The most common cause of biliary disease in patients with HIV is sclerosing cholangitis due to infections such as CMV, Cryptosporidium and Microsporidia

Pancreatitis in the context of HIV infection may be secondary to anti-retroviral treatment (especially didanosine) or by opportunistic infections e.g. CMV

IBD: histology

This histological differences between ulcerative colitis and Crohn's are summarised below:

Ulcerative colitis

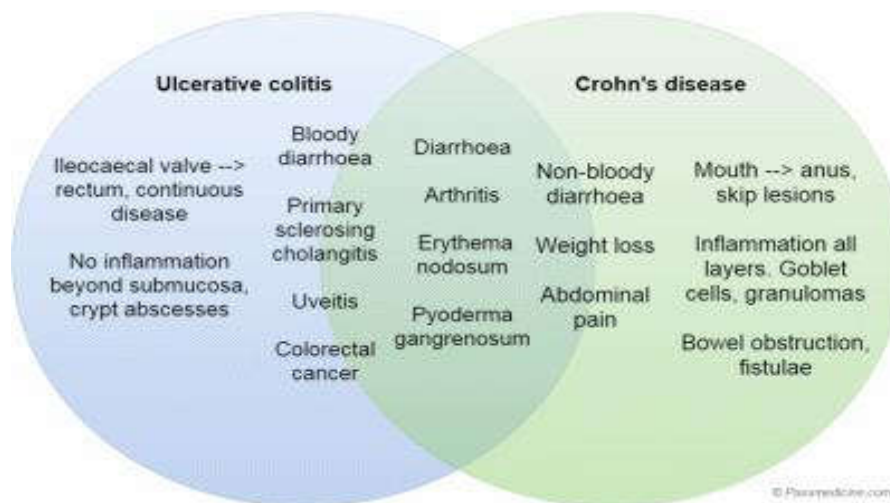
- inflammation in mucosa and submucosa only (unless fulminant disease)
- widespread ulceration with preservation of adjacent mucosa which has the appearance of polyps ('pseudopolyps')
- inflammatory cell infiltrate in lamina propria
- crypt abscesses
- depletion of goblet cells and mucin from gland epithelium
- granulomas are infrequent

Crohn's

- inflammation occurs in all layers, down to the serosa. This predisposes to strictures, fistulas and adhesions
- oedema of mucosa and submucosa, combined with deep fissured ulcers ('rose-thorn') leads to a 'cobblestone' pattern
- lymphoid aggregates
- non-caseating granulomas

Inflammatory bowel disease: key differences

The two main types of inflammatory bowel disease are Crohn's disease and Ulcerative colitis. They have many similarities in terms of presenting symptoms, investigation findings and management options.



Venn diagram showing shared features and differences between ulcerative colitis and Crohn's disease. Note that whilst some features are present in both, some are much more common in one of the conditions, for example colorectal cancer in ulcerative colitis

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There are however some key differences which are highlighted in table below:

	Crohn's disease (CD)	Ulcerative colitis (UC)
Features	Diarrhoea usually non-bloody Weight loss more prominent Upper gastrointestinal symptoms, mouth ulcers, perianal disease Abdominal mass palpable in the right iliac fossa	Bloody diarrhoea more common Abdominal pain in the left lower quadrant Tenesmus
Extra-intestinal	Gallstones are more common secondary to reduced bile acid reabsorption Oxalate renal stones*	Primary sclerosing cholangitis more common
Complications	Obstruction, fistula, colorectal cancer	Risk of colorectal cancer high in UC than CD
Pathology	Lesions may be seen anywhere from the mouth to anus Skip lesions may be present	Inflammation always starts at rectum and never spreads beyond ileocaecal valve Continuous disease
Histology	Inflammation in all layers from mucosa to serosa • increased goblet cells • granulomas	No inflammation beyond submucosa (unless fulminant disease) - inflammatory cell infiltrate in lamina propria • neutrophils migrate through the walls of glands to form crypt abscesses • depletion of goblet cells and mucin from gland epithelium • granulomas are infrequent
Endoscopy	Deep ulcers, skip lesions - 'cobble-stone' appearance	Widespread ulceration with preservation of adjacent mucosa which has the appearance of polyps ('pseudopolyps')
Radiology	<u>Small bowel enema</u> ▪ high sensitivity and specificity for examination of the terminal ileum ▪ strictures: 'Kantor's string sign' ▪ proximal bowel dilation ▪ 'rose thorn' ulcers ▪ fistulae	<u>Barium enema</u> ▪ loss of haustrations ▪ superficial ulceration, 'pseudopolyps' ▪ long standing disease: colon is narrow and short - 'drainpipe colon'

*impaired bile acid reabsorption increases the loss calcium in the bile. Calcium normally binds oxalate.

Inherited causes of jaundice

There are 4 inherited causes of jaundice you need to be aware of: Gilbert's syndrome, Crigler-Najjar syndrome, Dubin-Johnson syndrome and Rotor's syndrome. **It is important for the exam to be able to classify them according to whether they cause conjugated or unconjugated hyperbilirubinaemia:**

Unconjugated hyperbilirubinaemia	Conjugated hyperbilirubinaemia
Gilbert's syndrome Crigler-Najjar syndrome	Dubin-Johnson syndrome Rotor syndrome

Important points for the exam:

Gilbert's syndrome

- autosomal recessive
- UDP-glucuronyl transferase deficiency causing defective conjugation
- Benign

Crigler-Najjar syndrome, type 1

- autosomal recessive
- absolute deficiency of UDP-glucuronosyl transferase
- do not survive to adulthood

Crigler-Najjar syndrome, type 2

- slightly more common than type 1 and less severe
- may improve with phenobarbital

Dubin-Johnson syndrome

- autosomal recessive. Relatively common in Iranian Jews
- mutation in the canalicular multidrug resistance protein 2 (MRP2) results in defective hepatic excretion of bilirubin
- results in a grossly black liver
- benign

Rotor syndrome

- autosomal recessive
- defect in the hepatic uptake and storage of bilirubin
- benign

Iron studies

Serum iron

Total iron binding capacity (TIBC)

- transferrin
- raised in iron deficiency anaemia (IDA)
- raised in pregnancy and by oestrogen

Transferrin saturation

- calculated by serum iron / TIBC

Ferritin

- raised in inflammatory disorders
- low in IDA

Rarer tests

- transferrin receptors increased in IDA

Anaemia of chronic disease

- normochromic/hypochromic, normocytic anaemia
 - reduced serum and TIBC
 - normal or raised ferritin
-

Irritable bowel syndrome: diagnosis

NICE published clinical guidelines on the diagnosis and management of irritable bowel syndrome (IBS) in 2008

The diagnosis of IBS should be considered if the patient has had the following for at least 6 months:

- abdominal pain, and/or
- bloating, and/or
- change in bowel habit

A positive diagnosis of IBS should be made if the patient has abdominal pain relieved by defecation or associated with altered bowel frequency stool form, in addition to 2 of the following 4 symptoms:

- altered stool passage (straining, urgency, incomplete evacuation)
- abdominal bloating (more common in women than men), distension, tension or hardness
- symptoms made worse by eating
- passage of mucus

♦Features such as lethargy, nausea, backache and bladder symptoms may also support the diagnosis

Red flag features should be enquired about:

- rectal bleeding
- unexplained/unintentional weight loss
- family history of bowel or ovarian cancer
- onset after 60 years of age

Suggested primary care investigations are:

- full blood count
- ESR/CRP
- coeliac disease screen (tissue transglutaminase antibodies)

External Links

[NICE](#)

2015 IBS guidelines

Irritable bowel syndrome: management

The management of irritable bowel syndrome (IBS) is often difficult and varies considerably between patients. NICE updated it's guidelines in 2015.

First-line pharmacological treatment - according to predominant symptom

- pain: antispasmodic agents
- constipation: laxatives but avoid lactulose
- diarrhoea: loperamide is first-line

For patients with constipation who are not responding to conventional laxatives linaclotide may be considered, if:

- optimal or maximum tolerated doses of previous laxatives from different classes have not helped and
- they have had constipation for at least 12 months

Second-line pharmacological treatment

- low-dose tricyclic antidepressants (e.g. amitriptyline 5-10 mg) are used in preference to selective serotonin reuptake inhibitors

Other management options

- psychological interventions - if symptoms do not respond to pharmacological treatments after 12 months and who develop a continuing symptom profile (refractory IBS), consider referring for cognitive behavioural therapy, hypnotherapy or psychological therapy
- complementary and alternative medicines: 'do not encourage use of acupuncture or reflexology for the treatment of IBS'.

General dietary advice

- have regular meals and take time to eat
- avoid missing meals or leaving long gaps between eating
- drink at least 8 cups of fluid per day, especially water or other non-caffeinated drinks such as herbal teas
- restrict tea and coffee to 3 cups per day
- reduce intake of alcohol and fizzy drinks
- consider limiting intake of high-fibre food (for example, wholemeal or high-fibre flour and breads, cereals high in bran, and whole grains such as brown rice)
- reduce intake of 'resistant starch' often found in processed foods
- limit fresh fruit to 3 portions per day
- for diarrhoea, avoid sorbitol
- for wind and bloating consider increasing intake of oats (for example, oat-based breakfast cereal or porridge) and linseeds (up to one tablespoon per day).

External Links

[NICE](#)

2015 irritable bowel syndrome guidelines

[Kings College London](#)

Low FODMAP diet

Ischaemic hepatitis

Ischaemic hepatitis is a diffuse hepatic injury resulting from acute hypoperfusion (sometimes known as 'shock liver'). It is not an inflammatory process. It is diagnosed in the presence of an inciting event (in this case a cardiac arrest) and marked increases in aminotransferase levels (exceeding 1000 international unit/L or 50 times the upper limit of normal). Often, it will occur in conjunction with acute kidney injury (tubular necrosis) or other end organ dysfunction.

Jejunal villous atrophy

Whilst coeliac disease is the classic cause of jejunal villous atrophy there are a number of other causes you need to be aware of

Causes

- coeliac disease
- tropical sprue
- hypogammaglobulinaemia
- gastrointestinal lymphoma
- Whipple's disease
- cow's milk intolerance

Liver biopsy

Contraindications to percutaneous liver biopsy:

- deranged clotting (e.g. INR > 1.4)
- low platelets (e.g. < 60 * 10⁹/l)
- anaemia
- extrahepatic biliary obstruction
- hydatid cyst
- haemoangioma
- uncooperative patient
- ascites

External Links

[British Society of Gastroenterology](#)

2004 liver biopsy guidelines

Malabsorption

Malabsorption is characterised by diarrhoea, steatorrhoea and weight loss. Causes may be broadly divided into intestinal (e.g. villous atrophy), pancreatic (deficiency of pancreatic enzyme production or secretion) and biliary (deficiency of bile-salts needed for emulsification of fats)

Intestinal causes of malabsorption:

- coeliac disease
- Crohn's disease
- tropical sprue
- Whipple's disease
- Giardiasis
- brush border enzyme deficiencies (e.g. lactase insufficiency)

Pancreatic causes of malabsorption

- chronic pancreatitis
- cystic fibrosis
- pancreatic cancer

Biliary causes of malabsorption

- biliary obstruction
- primary biliary cirrhosis

Other causes:

- bacterial overgrowth (e.g. systemic sclerosis, diverticulae, blind loop)
- short bowel syndrome
- lymphoma

Malnutrition

Malnutrition is an important consequence of and contributor to chronic disease. It is clearly a complex and multifactorial problem that can be difficult to manage but there are a number of key points to remember for the exam.

NICE define malnutrition as the following:

- a Body Mass Index (BMI) of less than 18.5; or
- unintentional weight loss greater than 10% within the last 3-6 months; or
- a BMI of less than 20 and unintentional weight loss greater than 5% within the last 3-6 months

◆ Around 10% of patients aged over 65 years are malnourished, the vast majority of those living independently, i.e. not in hospital or care/nursing homes.

◆ Screening for malnutrition is mostly done using **MUST** (Malnutrition Universal Screen Tool). A link is provided to a copy of the MUST score algorithm.

- it should be done on admission to care/nursing homes and hospital, or if there is concern. For example an elderly, thin patient with pressure sores
- it takes into account BMI, recent weight change and the presence of acute disease
- categorises patients into low, medium and high risk

Management of malnutrition is difficult. NICE recommend the following points:

- dietician support if the patient is high-risk
- a 'food-first' approach with clear instructions (e.g. 'add full-fat cream to mashed potato'), rather than just prescribing oral nutritional supplements (ONS) such as Ensure
- if ONS are used they should be taken between meals, rather than instead of meals

External Links

[NICE](#)

2012 Quality Statement: Nutrition support in adults

[BAPEN](#)

MUST screening tool

Meckel's diverticulum

Meckel's diverticulum is a congenital diverticulum of the small intestine. It is a remnant of the omphalomesenteric duct (also called the vitellointestinal duct) and contains ectopic ileal, gastric or pancreatic mucosa

Rule of 2's:

- occurs in 2% of the population
- is 2 feet from the ileocaecal valve
- is 2 inches long

Presentation (usually asymptomatic)

- abdominal pain mimicking appendicitis
- rectal bleeding
- intestinal obstruction: secondary to an omphalomesenteric band (most commonly), volvulus and intussusception

Management

- removal if narrow neck or symptomatic. Options are between wedge excision or formal small bowel resection and anastomosis.

Pathophysiology

- normally, in the foetus, there is an attachment between the vitellointestinal duct and the yolk sac. This disappears at 6 weeks gestation
- the tip is free in the majority of cases
- associated with enterocystomas, umbilical sinuses, and omphaloileal fistulas.
- arterial supply: omphalomesenteric artery.
- typically lined by ileal mucosa but ectopic gastric mucosa can occur, with the risk of peptic ulceration. Pancreatic and jejunal mucosa can also occur.

Melanosis coli

Melanosis coli is a disorder of pigmentation of the bowel wall. Histology demonstrates pigment-laden macrophages

It is associated with laxative abuse, especially anthraquinone compounds such as senna

Mesenteric ischaemia

Mesenteric ischaemia is primarily caused by arterial embolism resulting in infarction of the colon. It is more likely to occur in areas such as the splenic flexure that are located at the borders of the territory supplied by the superior and inferior mesenteric arteries.

Predisposing factors:

- increasing age
- atrial fibrillation
- other causes of emboli: endocarditis
- cardiovascular disease risk factors: smoking, hypertension, diabetes
- cocaine: ischaemic colitis is sometimes seen in young patients following cocaine use

Features

- abdominal pain
- rectal bleeding
- diarrhoea
- fever
- bloods typically show an elevated WBC associated with acidosis

Management

- supportive care
- laparotomy and bowel resection

Metoclopramide:

Metoclopramide is a D2 receptor antagonist mainly used in the management of nausea. Other uses include:

- gastro-oesophageal reflux disease
- prokinetic action is useful in gastroparesis secondary to diabetic neuropathy
- often combined with analgesics for the treatment of migraine (migraine attacks result in gastroparesis, slowing the absorption of analgesics)

Adverse effects

- extrapyramidal effects: oculogyric crisis. This is particularly a problem in children and young adults
- hyperprolactinaemia
- tardive dyskinesia
- parkinsonism

Non-alcoholic fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) is now the most common cause of liver disease in the developed world. It is largely caused by obesity and describes a spectrum of disease ranging from:

- steatosis - fat in the liver
- steatohepatitis - fat with inflammation, non-alcoholic steatohepatitis (NASH), see below
- progressive disease may cause fibrosis and liver cirrhosis

◆NAFLD is thought to represent the hepatic manifestation of the metabolic syndrome and hence insulin resistance is thought to be the key mechanism leading to steatosis

◆Non-alcoholic steatohepatitis (NASH) is a term used to describe liver changes similar to those seen in alcoholic hepatitis in the absence of a history of alcohol abuse. It is relatively common and thought to affect around 3-4% of the general population. The progression of disease in patients with NASH may be responsible for a proportion of patients previously labelled as cryptogenic cirrhosis.

Associated factors:

- obesity
- hyperlipidaemia
- type 2 diabetes mellitus
- jejunoileal bypass
- sudden weight loss/starvation

Features

- usually asymptomatic
- hepatomegaly
- ALT is typically greater than AST
- increased echogenicity on ultrasound

Management

- the mainstay of treatment is lifestyle changes (particularly weight loss) and monitoring
- there is ongoing research into the role of gastric banding and insulin-sensitising drugs (e.g. Metformin)

Oesophageal disorders

The table below lists a small group of oesophageal disorders that are not covered elsewhere in the notes.

Disorder	Notes
Plummer-Vinson syndrome	Triad of: • dysphagia (secondary to oesophageal webs) • glossitis • iron-deficiency anaemia Treatment includes iron supplementation and dilation of the webs
Mallory-Weiss syndrome	Severe vomiting → painful mucosal lacerations at the gastroesophageal junction resulting in haematemesis. Common in alcoholics
Boerhaave syndrome	Severe vomiting → oesophageal rupture

Esophageal varices

Acute treatment of variceal hemorrhage:

- ABC: patients should ideally be resuscitated prior to endoscopy
- correct clotting: FFP, vitamin K
- vasoactive agents: terlipressin is currently the only licensed vasoactive agent and is supported by NICE guidelines. It has been shown to be of benefit in initial haemostasis and preventing rebleeding. Octreotide may also be used although there is some evidence that terlipressin has a greater effect on reducing mortality
- prophylactic antibiotics have been shown in multiple meta-analyses to reduce mortality in patients with liver cirrhosis. Quinolones are typically used.
- endoscopy: endoscopic variceal band ligation is superior to endoscopic sclerotherapy. NICE recommend band ligation
- Sengstaken-Blakemore tube if uncontrolled haemorrhage
- Transjugular Intrahepatic Portosystemic Shunt (TIPSS) if above measures fail

Prophylaxis of variceal haemorrhage

- propranolol: reduced rebleeding and mortality compared to placebo
- endoscopic variceal band ligation (EVL) is superior to endoscopic sclerotherapy. It should be performed at two-weekly intervals until all varices have been eradicated. Proton pump inhibitor cover is given to prevent EVL-induced ulceration

External Links

[NICE](#)

2012 Acute upper gastrointestinal bleeding: management

[Royal College of Physicians](#)

2012 Managing acute upper gastrointestinal bleeding in the acute assessment unit

[British Society of Gastroenterology](#)

Management of oesophageal varices

[Cochrane](#)

Antibiotic prophylaxis for cirrhotic patients with gastrointestinal bleeding

Pancreatic cancer

Pancreatic cancer is often diagnosed late as it tends to present in a non-specific way. Over 80% of pancreatic tumours are adenocarcinomas which typically occur at the head of the pancreas.

Associations

- increasing age
- smoking
- diabetes
- chronic pancreatitis (alcohol does not appear an independent risk factor though)
- hereditary non-polyposis colorectal carcinoma
- multiple endocrine neoplasia
- BRCA2 gene

Features

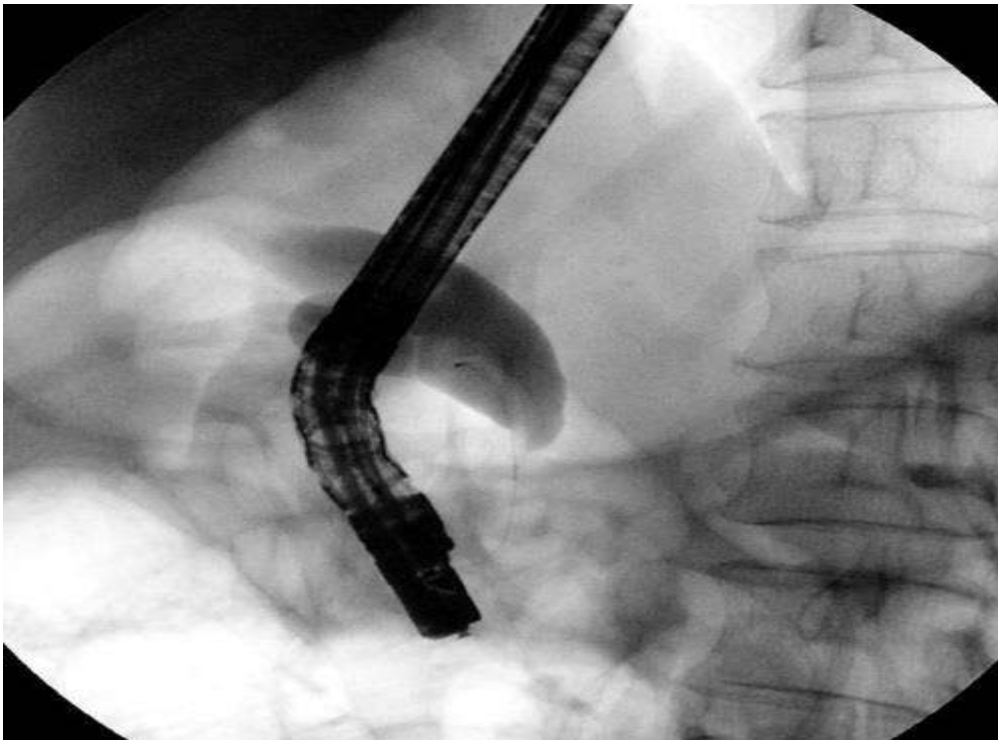
- classically painless jaundice
- however, patients typically present in a non-specific way with anorexia, weight loss, epigastric pain
- loss of exocrine function (e.g. steatorrhoea)
- atypical back pain is often seen
- migratory thrombophlebitis (Trousseau sign) is more common than with other cancers

Investigation

- ultrasound has a sensitivity of around 60-90%
- high resolution CT scanning is the investigation of choice if the diagnosis is suspected

Management

- less than 20% are suitable for surgery at diagnosis
- a Whipple's resection (pancreaticoduodenectomy) is performed for resectable lesions in the head of pancreas. Side-effects of a Whipple's include dumping syndrome and peptic ulcer disease
- adjuvant chemotherapy is usually given following surgery
- ERCP with stenting is often used for palliation



© Image used on license from [Radiopaedia](#)

ERCP showing invasive ductal adenocarcinoma. Note the dilation of the common bile duct due to the pancreatic lesion

Pancreatic cancer: features and investigation

Pancreatic cancer is often diagnosed late as it tends to present in a non-specific way. Over 80% of pancreatic tumours are adenocarcinomas which typically occur at the head of the pancreas.

Associations

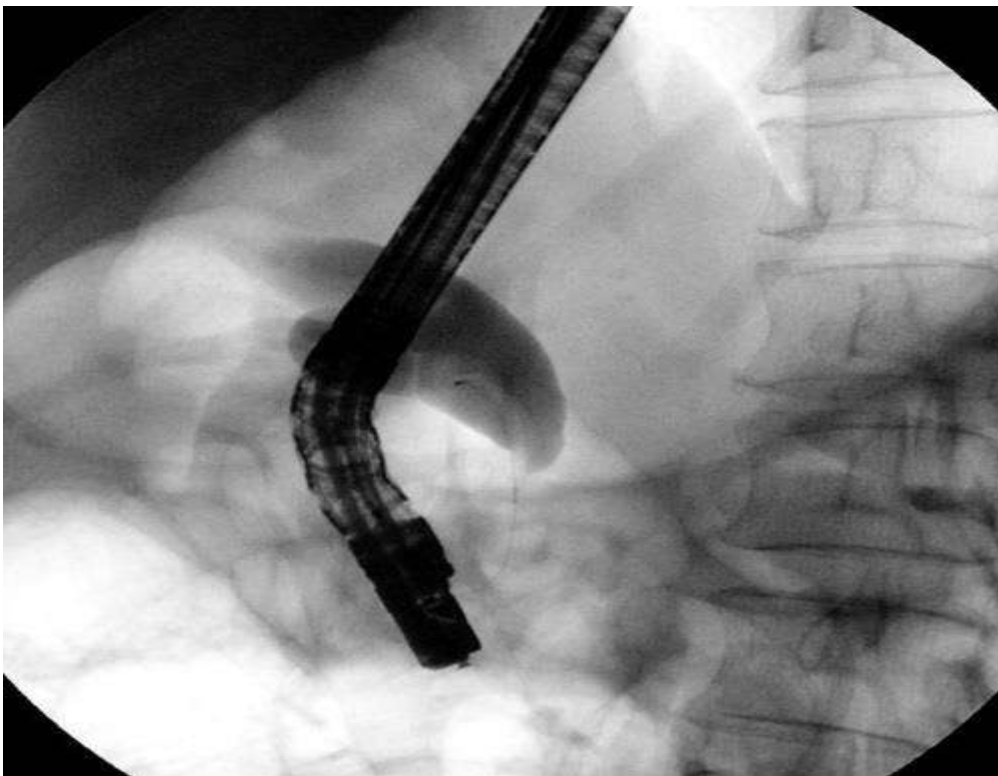
- increasing age
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ERCP showing invasive ductal adenocarcinoma. Note the dilation of the common bile duct due to the pancreatic lesion

Pernicious anaemia: investigation

Investigation

- anti gastric parietal cell antibodies in 90% (but low specificity)
- anti intrinsic factor antibodies in 50% (specific for pernicious anaemia)
- macrocytic anaemia
- low WCC and platelets
- LDH may be raised due to ineffective erythropoiesis
- also low serum B12, hypersegmented polymorphs on film, megaloblasts in marrow
- Schilling test

Schilling test

- radiolabelled B12 given on two occasions
- first on its own
- second with oral IF
- urine B12 levels measured

Peutz-Jeghers syndrome

Peutz-Jeghers syndrome is an autosomal dominant condition characterised by numerous hamartomatous polyps in the gastrointestinal tract. It is also associated with pigmented freckles on the lips, face, palms and soles. Around 50% of patients will have died from a gastrointestinal tract cancer by the age of 60 years.

Genetics

- autosomal dominant
- responsible gene encodes serine threonine kinase LKB1 or STK11

Features

- hamartomatous polyps in GI tract (mainly small bowel)
- pigmented lesions on lips, oral mucosa, face, palms and soles
- intestinal obstruction e.g. intussusception
- gastrointestinal bleeding

Management

- conservative unless complications develop

External Links

[DermIS](#)

Peutz-Jeghers syndrome images

Pharyngeal pouch

A pharyngeal pouch is a posteromedial diverticulum through Killian's dehiscence. Killian's dehiscence is a triangular area in the wall of the pharynx between the thyropharyngeus and cricopharyngeus muscles. It is more common in older patients and is 5 times more common in men

Features

- dysphagia
- regurgitation
- aspiration
- neck swelling which gurgles on palpation
- halitosis



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Still image taken from a barium swallow with fluoroscopy. During swallowing an outpouching of the posterior hypopharyngeal wall is visualised at the level C5-C6, right above the upper oesophageal sphincter

Post-cholecystectomy syndrome

◆Post-cholecystectomy syndrome is a recognised complication of cholecystectomies. Typically symptoms of dyspepsia, vomiting, pain, flatulence and diarrhoea occur in up to 40% patients post surgery.

◆The pathology behind the syndrome isn't completely clear, however there is some association with remnant stones and biliary injury. Pain is often due to sphincter of Oddi dysfunction and the development of surgical adhesions.

◆Management is often difficult, but often involves a low-fat diet and the introduction of bile acid sequestrants, such as Cholestyramine, to bind the excess bile acids and thus preventing lower gastrointestinal signs. Proton-pump inhibitors like Lansoprazole do play a role, if the patient is complaining of dyspeptic like symptoms. Antibiotics and pancreatic enzyme replacements play no part in management.

Pregnancy: jaundice

Intrahepatic cholestasis of pregnancy

Intrahepatic cholestasis of pregnancy (also known as obstetric cholestasis) occurs in around 1% of pregnancies and is generally seen in the third trimester. It is the most common liver disease of pregnancy.

Features

- pruritus, often in the palms and soles
- no rash (although skin changes may be seen due to scratching)
- raised bilirubin

Management

- ursodeoxycholic acid is used for symptomatic relief
- women are typically induced at 37 weeks

Complications include an increased rate of stillbirth. It is not generally associated with increased maternal morbidity.

Acute fatty liver of pregnancy

Acute fatty liver of pregnancy is rare complication which may occur in the third trimester or the period immediately following delivery.

Features

- abdominal pain
- nausea & vomiting
- headache
- jaundice
- hypoglycaemia
- severe disease may result in pre-eclampsia

Investigations

- ALT is typically elevated e.g. 500 u/l

Management

- support care
- once stabilised delivery is the definitive management

♦Gilbert's, Dubin-Johnson syndrome, may be exacerbated during pregnancy

HELLP

- Haemolysis, Elevated Liver enzymes, Low Platelets

External Links

[Canadian Journal of Gastroenterology](#)

Acute Fatty Liver of Pregnancy

Primary biliary cirrhosis

Primary biliary cirrhosis (now increasingly referred to as primary biliary cholangitis) is a chronic liver disorder typically seen in middle-aged females (female:male ratio of 9:1). The aetiology is not fully understood although it is thought to be an autoimmune condition. Interlobular bile ducts become damaged by a chronic inflammatory process causing progressive cholestasis which may eventually progress to cirrhosis. The classic presentation is itching in a middle-aged woman

Associations

- Sjogren's syndrome (seen in up to 80% of patients)
- rheumatoid arthritis
- systemic sclerosis
- thyroid disease

Diagnosis

- anti-mitochondrial antibodies (AMA) M2 subtype are present in 98% of patients and are highly specific
- smooth muscle antibodies in 30% of patients
- raised serum IgM

Management

- pruritus: cholestyramine
- fat-soluble vitamin supplementation
- ursodeoxycholic acid
- liver transplantation e.g. if bilirubin > 100 (PBC is a major indication) - recurrence in graft can occur but is not usually a problem

Primary biliary cirrhosis: features

Primary biliary cirrhosis (now increasingly referred to as primary biliary cholangitis) is a chronic liver disorder typically seen in middle-aged females (female:male ratio of 9:1). The aetiology is not fully understood although it is thought to be an autoimmune condition. Interlobular bile ducts become damaged by a chronic inflammatory process causing progressive cholestasis, which may eventually progress to cirrhosis. The classic presentation is itching in a middle-aged woman

Clinical features:

- early: may be asymptomatic (e.g. raised ALP on routine LFTs) or fatigue, pruritus
- cholestatic jaundice
- hyperpigmentation, especially over pressure points
- xanthelasmas, xanthomata
- also: clubbing, hepatosplenomegaly
- late: may progress to liver failure

Complications:

- malabsorption: osteomalacia, coagulopathy
- sicca syndrome occurs in 70% of cases
- portal hypertension: ascites, variceal haemorrhage
- hepatocellular cancer (20-fold increased risk)

Primary sclerosing cholangitis

Primary sclerosing cholangitis is a biliary disease of unknown aetiology characterised by inflammation and fibrosis of intra and extra-hepatic bile ducts

Associations

- ulcerative colitis: 4% of patients with UC have PSC, 80% of patients with PSC have UC
- Crohn's (much less common association than UC)
- HIV

Features

- cholestasis: jaundice and pruritus
- right upper quadrant pain
- fatigue

Investigation

- ERCP is the standard diagnostic tool, showing multiple biliary strictures giving a 'beaded' appearance
- ANCA may be positive
- there is a limited role for liver biopsy, which may show fibrous, obliterative cholangitis often described as 'onion skin'

Complications

- cholangiocarcinoma (in 10%)
- increased risk of colorectal cancer

Proton pump inhibitors

Proton pump inhibitors (PPI) are a group of drugs which profoundly reduce acid secretion in the stomach. They irreversibly block the hydrogen/potassium adenosine triphosphatase enzyme system (the H^+/K^+ ATPase) of the gastric parietal cell

Examples include omeprazole and lansoprazole

Pyloric stenosis

Pyloric stenosis typically presents in the second to fourth weeks of life with vomiting, although rarely may present later at up to four months. It is caused by hypertrophy of the circular muscles of the pylorus

Epidemiology

- incidence of 4 per 1,000 live births
- 4 times more common in males
- 10-15% of infants have a positive family history
- first-borns are more commonly affected

Features

- 'projectile' vomiting, typically 30 minutes after a feed
- constipation and dehydration may also be present
- a palpable mass may be present in the upper abdomen
- hypochloraemic, hypokalaemic alkalosis due to persistent vomiting

Diagnosis is most commonly made by ultrasound

Management is with Ramstedt pyloromyotomy

Refeeding syndrome

Refeeding syndrome describes the metabolic abnormalities which occur on feeding a person following a period of starvation. **The metabolic consequences include:**

- hypophosphataemia
- hypokalaemia
- hypomagnesaemia
- abnormal fluid balance

These abnormalities can lead to organ failure.

Prevention

NICE produced guidelines in 2006 on nutritional support. Refeeding syndrome may be avoided by identifying patients at a high-risk of developing refeeding syndrome:

Patients are considered high-risk if one or more of the following:

- BMI < 16 kg/m²
- unintentional weight loss >15% over 3-6 months
- little nutritional intake > 10 days
- hypokalaemia, hypophosphataemia or hypomagnesaemia prior to feeding (unless high)

If two or more of the following:

- BMI < 18.5 kg/m²
- unintentional weight loss > 10% over 3-6 months
- little nutritional intake > 5 days
- history of: alcohol abuse, drug therapy including insulin, chemotherapy, diuretics and antacids

NICE recommend that if a patient hasn't eaten for > 5 days, aim to re-feed at no more than 50% of requirements for the first 2 days.

External Links

[NICE](#)

2006 Nutrition support in adults

Skin disorders associated with malignancy

Paraneoplastic syndromes associated with internal malignancies:

Skin disorder	Associated malignancies
Acanthosis nigricans	Gastric cancer
Acquired ichthyosis	Lymphoma
Acquired hypertrichosis lanuginosa	Gastrointestinal and lung cancer
Dermatomyositis	Ovarian and lung cancer
Erythema gyratum repens	Lung cancer
Erythroderma	Lymphoma
Migratory thrombophlebitis	Pancreatic cancer
Necrolytic migratory erythema	Glucagonoma
Pyoderma gangrenosum (bullous and non-bullous forms)	Myeloproliferative disorders
Sweet's syndrome	Haematological malignancy e.g. Myelodysplasia - tender, purple plaques
Tylosis	Oesophageal cancer

External Links

[DermNet NZ](#)

Acanthosis nigricans

[DermNet NZ](#)

Dermatomyositis

[DermNet NZ](#)

Pyoderma gangrenosum

[DermNet NZ](#)

Ichthyosis

Small bowel bacterial overgrowth syndrome

Small bowel bacterial overgrowth syndrome (SBBOS) is a disorder characterised by excessive amounts of bacteria in the small bowel resulting in gastrointestinal symptoms.

Risk factors for SBBOS

- neonates with congenital gastrointestinal abnormalities
- scleroderma
- diabetes mellitus

It should be noted that many of the features overlap with irritable bowel syndrome:

- chronic diarrhoea
- bloating, flatulence
- abdominal pain

Diagnosis

- hydrogen breath test

Management

- correction of underlying disorder
 - antibiotic therapy: rifaximin is now the treatment of choice due to relatively low resistance. Co-amoxiclav or metronidazole are also effective in the majority of patients.
-

Spironolactone

Spironolactone is an aldosterone antagonist which acts in the cortical collecting duct.

Indications

- ascites: patients with cirrhosis develop a secondary hyperaldosteronism. Relatively large doses such as 100 or 200mg are often used
- hypertension: used in some patients as a NICE 'step 4' treatment
- heart failure (see RALES study below)
- nephrotic syndrome
- Conn's syndrome

Adverse effects

- hyperkalaemia
- gynaecomastia

RALES

- NYHA III + IV, patients already taking ACE inhibitor
- low dose spironolactone reduces all cause mortality

Spontaneous bacterial peritonitis

Spontaneous bacterial peritonitis (SBP) is a form of peritonitis usually seen in patients with ascites secondary to liver cirrhosis.

Diagnosis

- paracentesis: neutrophil count > 250 cells/ul

Management

- intravenous cefotaxime is usually given

Antibiotic prophylaxis should be given if:

- patients who have had an episode of SBP
- patients with fluid protein <15 g/l and either Child-Pugh score of at least 9 or hepatorenal syndrome

Alcoholic liver disease is a marker of poor prognosis in SBP.

External Links

[British Society of Gastroenterology](#)

Guidelines on the management of ascites in cirrhosis

Total parenteral nutrition

- Commonly used in nutritionally compromised surgical patients
- Bags contain combinations of glucose, lipids and essential electrolytes, the exact composition is determined by the patients nutritional requirements.
- Although it may be infused peripherally, this may result in thrombophlebitis.
- Longer term infusions should be administered into a central vein (preferably via a PICC line).
- Complications are related to sepsis, re-feeding syndromes and hepatic dysfunction.

Ulcerative colitis

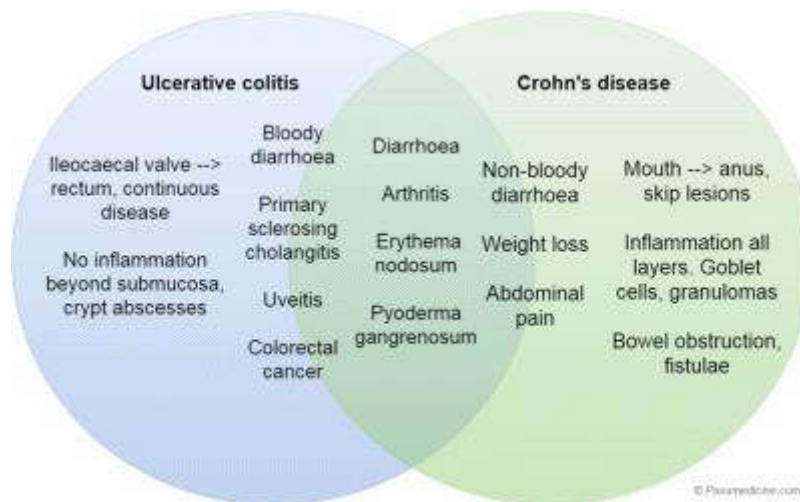
Ulcerative colitis (UC) is a form of inflammatory bowel disease. Inflammation always starts at rectum (hence it is the most common site for UC), never spreads beyond ileocaecal valve and is continuous. The peak incidence of ulcerative colitis is in people aged 15-25 years and in those aged 55-65 years.

The initial presentation is usually following insidious and intermittent symptoms. Features include:

- bloody diarrhoea
- urgency
- tenesmus
- abdominal pain, particularly in the left lower quadrant
- extra-intestinal features (see below).

Questions regarding the 'extra-intestinal' features of inflammatory bowel disease are common:

	Common to both Crohn's disease (CD) and Ulcerative colitis (UC)	Notes
Related to disease activity	Arthritis: pauciarticular, asymmetric Erythema nodosum Episcleritis Osteoporosis	Arthritis is the most common extra-intestinal feature in both CD and UC Episcleritis is more common in CD
Unrelated to disease activity	▪Arthritis: polyarticular, symmetric ▪Uveitis ▪Pyoderma gangrenosum ▪Clubbing ▪Primary sclerosing cholangitis	▪Primary sclerosing cholangitis is much more common in UC ▪Uveitis is more common in UC



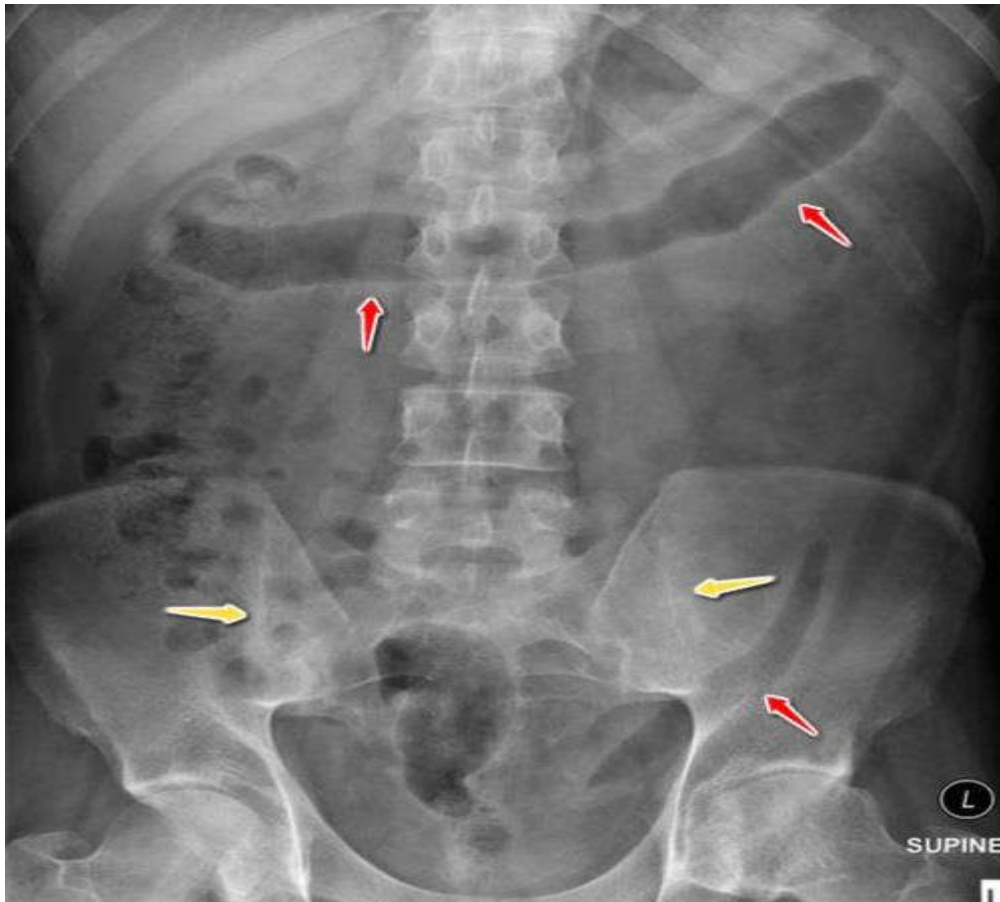
Venn diagram showing shared features and differences between ulcerative colitis and Crohn's disease. Note that whilst some features are present in both, some are much more common in one of the conditions, for example colorectal cancer in ulcerative colitis.

Pathology:

- red, raw mucosa, bleeds easily
- no inflammation beyond submucosa (unless fulminant disease)
- widespread ulceration with preservation of adjacent mucosa which has the appearance of polyps ('pseudopolyps')
- inflammatory cell infiltrate in lamina propria
- neutrophils migrate through the walls of glands to form crypt abscesses
- depletion of goblet cells and mucin from gland epithelium
- granulomas are infrequent

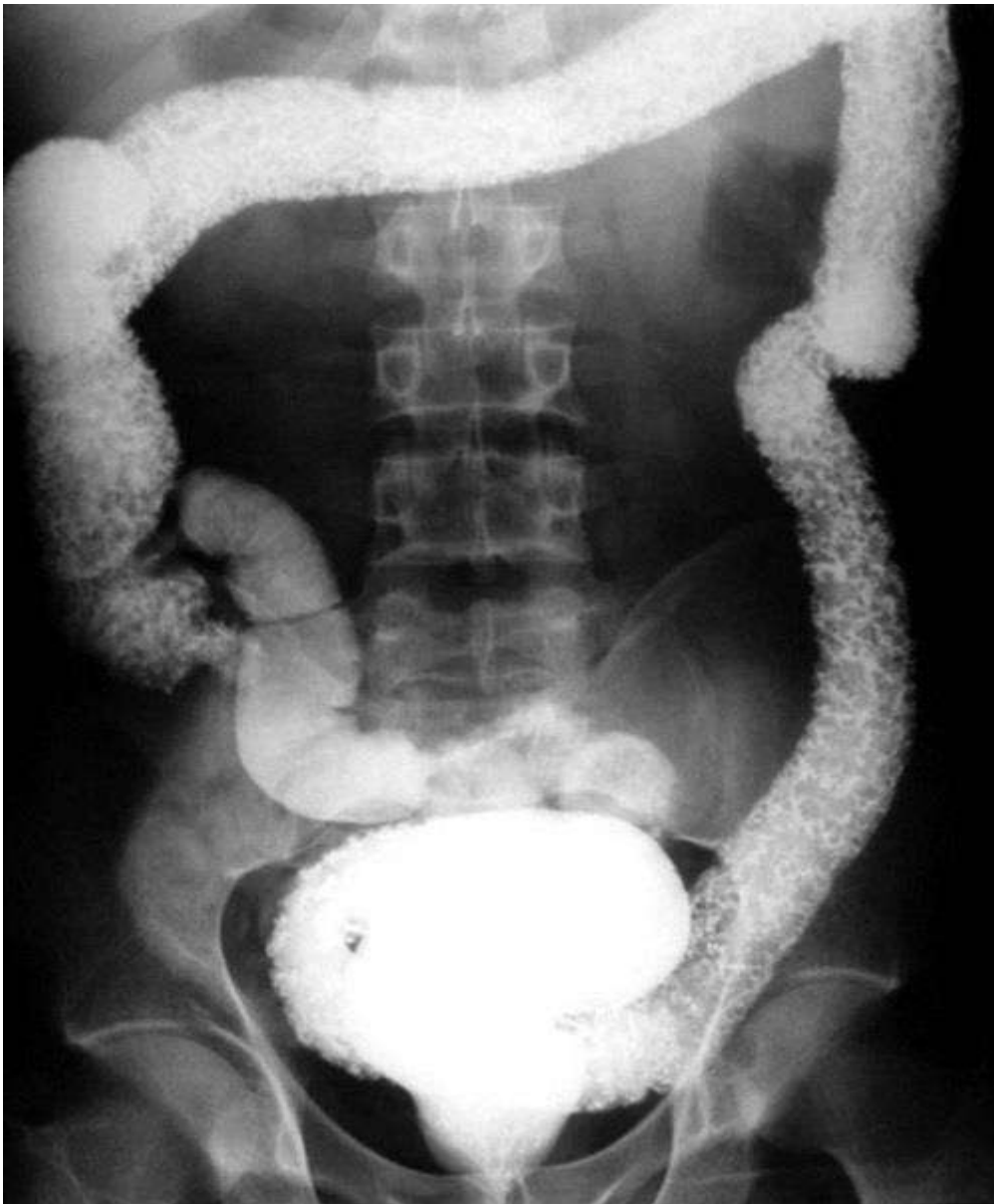
Barium enema:

- loss of haustrations
- superficial ulceration, 'pseudopolyps'
- long standing disease: colon is narrow and short -'drainpipe colon'



© Image used on license from [Radiopaedia](#)

Abdominal x-ray from a patient with ulcerative colitis showing lead pipe appearance of the colon (red arrows). Ankylosis of the left sacroiliac joint and partial ankylosis on the right (yellow arrow), reinforcing the link with sacroilitis.



© Image used on license from [Radiopaedia](#)



Barium enema from a patient with ulcerative colitis. The whole colon, without skips is affected by an irregular mucosa with loss of normal haustral markings.

External Links

[British Society of Gastroenterology](#)

2004 IBD guidelines

Ulcerative colitis: colorectal cancer

Overview

- risk of colorectal cancer is significantly higher than that of the general population although studies report widely varying rates
- the increased risk is mainly related to chronic inflammation
- worse prognosis than patients without ulcerative colitis (partly due to delayed diagnosis)
- lesions may be multifocal

Factors increasing risk of cancer

- disease duration > 10 years
- patients with pancolitis
- onset before 15 years old
- unremitting disease
- poor compliance to treatment

Colonoscopy surveillance in inflammatory bowel disease patients should be decided following risk stratification.

Lower risk

5 year follow up colonoscopy

- Extensive colitis with no active endoscopic/histological inflammation
- OR left sided colitis
- OR Crohn's colitis of <50% colon

Intermediate risk

3 year colonoscopy

- Extensive colitis with mild active endoscopy/histological inflammation
- OR post-inflammatory polyps
- OR family history of colorectal cancer in a first degree relative aged 50 or over.

Higher risk

1 year follow up colonoscopy:

- Extensive colitis with moderate/severe active endoscopic/histological inflammation
- OR stricture in past 5 years
- OR dysplasia in past 5 years declining surgery
- OR primary sclerosing cholangitis / transplant for primary sclerosing cholangitis
- OR family history of colorectal cancer in first degree relatives aged <50 years

External Links

[British Society of Gastroenterology](#)

2004 IBD guidelines

Ulcerative colitis: management

Treatment can be divided into inducing and maintaining remission. NICE released guidelines on the management of ulcerative colitis in 2013.

The severity of UC is usually classified as being mild, moderate or severe:

- mild: < 4 stools/day, only a small amount of blood
- moderate: 4-6 stools/day, varying amounts of blood, no systemic upset
- severe: >6 bloody stools per day + features of systemic upset (pyrexia, tachycardia, anaemia, raised inflammatory markers)

Inducing remission

- treatment depends on the extent and severity of disease
- rectal (topical) aminosalicylates or steroids: for distal colitis rectal mesalazine has been shown to be superior to rectal steroids and oral aminosalicylates
- oral aminosalicylates
- oral prednisolone is usually used second-line for patients who fail to respond to aminosalicylates. NICE recommend waiting around 4 weeks before deciding if first-line treatment has failed
- severe colitis should be treated in hospital. Intravenous steroids are usually given first-line

Maintaining remission

- oral aminosalicylates e.g. mesalazine
- azathioprine and mercaptopurine
- methotrexate is not recommended for the management of UC (in contrast to Crohn's disease)
- there is some evidence that probiotics may prevent relapse in patients with mild to moderate disease

External Link

[NICE](#)

2013 Ulcerative colitis guidelines

[British Society of Gastroenterology](#)

2004 IBD guidelines

Villous adenoma

Villous adenomas are colonic polyps with the potential for malignant transformation. They characteristically secrete large amounts of mucous, potentially resulting in electrolyte disturbances.

The vast majority are asymptomatic. Possible features:

- non-specific lower gastrointestinal symptoms
- secretory diarrhoea may occur
- microcytic anaemia
- hypokalaemia

VIPoma

VIP (vasoactive intestinal peptide)

- source: small intestine, pancreas
- stimulation: neural
- actions: stimulates secretion by pancreas and intestines, inhibits acid and pepsinogen secretion

VIPoma

- 90% arise from pancreas
- large volume diarrhoea
- weight loss
- dehydration
- hypokalaemia, hypochlorhydria

Whipple's disease

Whipple's disease is a rare multi-system disorder caused by *Tropheryma whippelii* infection. It is more common in those who are HLA-B27 positive and in middle-aged men

Features

- malabsorption: diarrhoea, weight loss
- large-joint arthralgia
- lymphadenopathy
- skin: hyperpigmentation and photosensitivity
- pleurisy, pericarditis
- neurological symptoms (rare): ophthalmoplegia, dementia, seizures, ataxia, myoclonus.

Investigation

- jejunal biopsy shows deposition of macrophages containing Periodic acid-Schiff (PAS) granules

Management

- guidelines vary: oral co-trimoxazole for a year is thought to have the lowest relapse rate, sometimes preceded by a course of IV penicillin

Wilson's disease

Wilson's disease is an autosomal recessive disorder characterised by excessive copper deposition in the tissues. Metabolic abnormalities include increased copper absorption from the small intestine and decreased hepatic copper excretion. Wilson's disease is caused by a defect in the ATP7B gene located on chromosome 13.

The onset of symptoms is usually between 10 - 25 years. Children usually present with liver disease whereas the first sign of disease in young adults is often neurological disease

Features result from excessive copper deposition in the tissues, especially the brain, liver and cornea:

- liver: hepatitis, cirrhosis
- neurological: basal ganglia degeneration, speech and behavioural problems are often the first manifestations. Also: asterixis, chorea, dementia
- Kayser-Fleischer rings
- renal tubular acidosis (esp. Fanconi syndrome)
- haemolysis
- blue nails

Diagnosis

- reduced serum caeruloplasmin
- reduced serum copper (counter-intuitive, but 95% of plasma copper is carried by caeruloplasmin)
- increased 24hr urinary copper excretion

Management

- penicillamine (chelates copper) has been the traditional first-line treatment
 - trientine hydrochloride is an alternative chelating agent which may become first-line treatment in the future
 - tetrathiomolybdate is a newer agent that is currently under investigation
-

Zollinger-Ellison syndrome

Zollinger-Ellison syndrome is condition characterised by excessive levels of gastrin, usually from a gastrin secreting tumour usually of the duodenum or pancreas. Around 30% occur as part of MEN type I syndrome

Features

- multiple gastroduodenal ulcers
- diarrhoea
- malabsorption

Diagnosis

- fasting gastrin levels: the single best screen test
 - secretin stimulation test
-

External Links

[Patient.info](#)

Zollinger-Ellison syndrome